Abnormal somatosensory processing in schizophrenia: Changes in somatosensory sensitivity on a shortened version of the Kinaesthetic Figural Aftereffects task

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Declaration

A research report submitted in partial fulfillment of the requirements for the degree of Master of Arts (Research Psychology) in the Faculty of Humanities, University of the Witwatersrand, Johannesburg, 17 August 2010.

I declare that this research report is my own, unaided work. It has not been submitted before for any other degree or examination at this or any other university.

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Introduction

Approximately one percent of the world's population has schizophrenia, with one percent of the South African population affected by this disorder (Trump & Hugo, 2006). Schizophrenia is a worldwide public health problem, which results in significant economic and personal costs (Sadock & Sadock, 2004). It causes numerous problems for the sufferer, including discrimination and stigma from people who do not understand it. Up-to-date treatment is often expensive and inaccessible to many, and individuals with severe symptoms are often unable to work and support themselves, placing pressure on their families and communities (Trump & Hugo, 2006). In addition, there are limits on the amount of assistance that mentally ill individuals can obtain from their medical insurance and from the government through the public health system. Therefore, a great amount of research is directed towards understanding the aetiology of schizophrenia, as well as associated risk factors, in order to find more effective ways of dealing with and assisting the mentally ill, as well as early identification and in some cases, prevention.

Prior to discussing the aetiology of schizophrenia, it needs to be clearly defined and its prevalence and significance within the South African context discussed, which will be provided shortly. Following this, the various functional abnormalities that have been found to be present in individuals with schizophrenia will be discussed according to the manner in which these abnormalities have been examined, namely through fMRI, EEG, PET and behavioural measures. The behavioural measure utilized in this study, namely the Kinaesthetic Figural Aftereffects task (KFA), will then be discussed, after

which an overview of the various explanatory paradigms that have been put forward to make sense of these functional abnormalities, will be provided.

Schizophrenia: Definition and Diagnostics

The International Statistical Classification of Diseases and Related Health Problems [ICD 10] (2007) defines schizophrenia as a disorder characterized by fundamental and distinctive distortions of thinking and perception, as well as inappropriate and flat affect. It is noted that although cognitive deficits may develop over time, clear consciousness and intellectual capacity are usually sustained. A diagnosis of schizophrenia requires that two or more positive, negative and/or disorganized symptoms are present for a significant portion of time within a one-month period (American Psychiatric Association [APA], 2000).

Positive symptoms generally include a distortion of normal behaviour, including hallucinations and delusions. Hallucinations are defined as the experience of sensory events (either visual, tactile, olfactory, auditory or gustatory although auditory hallucinations are the most common) in the absence of any sensory stimulus originating from the surrounding environment, while delusions are incorrect beliefs that usually result from a misinterpretation of perceptions or experiences, and that prove exceptionally resistant to alteration (APA, 2000). Negative symptoms are characterized by deficits in normal behaviour, such as deficits in speech or motivation, including alogia or absence of speech, avolition or the inability to initiate and endure in activities, anhedonia or a lack of pleasure, and affective flattening where individuals speak in a flat, toneless manner and lack facial expressions and emotional displays. Lastly, disorganized symptoms include inappropriate affect, for example, laughing upon receiving bad news, incoherent speech or a lack of insight and jumping across

topics and irregular behaviour, such as disorganized or catatonic behaviour where individuals either act in a strange way or vary from complete immobility to excessive agitation, termed catatonia (APA, 2000).

As mentioned previously, two of these symptoms must be present for about one month in order to diagnose schizophrenia, with continuous signs of disturbance persisting for at least six months. In addition, the individual must exhibit marked social or occupational dysfunction not better accounted for by Schizoaffective Disorder or Mood Disorder with Psychotic Features and their condition not being a result of the direct physiological effects of a substance or general medical condition. Further, if an individual has previously been diagnosed with Autistic Disorder or another Pervasive Developmental Disorder, a diagnosis of schizophrenia can only be made if major hallucinations or delusions are also present for at least one month (APA, 2000). The ICD 10 (2007) adds that schizophrenia should not be diagnosed if a person is experiencing extensive depressive or manic symptoms, unless the schizophrenic symptoms precede these, as well as if a person is intoxicated or withdrawing from a drug. Therefore, the diagnosis of schizophrenia depends on an individual's history of illness and a comprehensive mental status examination, with no reliable laboratory tests having yet been developed (Hales, Yudofsky & Gabbard, 2008).

There are five subtypes of schizophrenia, namely the disorganized type, paranoid type, undifferentiated type, catatonic type and residual type, each with different prognoses and features (APA, 2000). Individuals suffering from the disorder may experience difficulties with daily goal-directed behaviours or activities, such as maintaining personal hygiene or preparing meals.

Prevalence

As mentioned previously, schizophrenia affects approximately one percent of the world's population and occurs in all societies and geographical areas. The incidence of schizophrenia is slightly higher in men than in women and is higher in urban areas than in rural areas, with the risk of schizophrenia related to the extent of urbanisation (Sadock & Sadock, 2004). In addition, schizophrenia tends to be more severe in high-income countries than in middle and low-income countries. Men tend to have an earlier onset than women, with the onset of the disorder occurring between the ages of 15 and 35, with the onset of schizophrenia before adolescence and after 50-years of age being very rare (Kaplan, Sadock & Grebb, 1994). A new case of schizophrenia arises in one out of every 10,000 people every year (APA, 2000). Recent immigrants have an increased risk of developing the disorder.

Schizophrenia causes significant and chronic impairments, although its course varies between being chronic and having aggravated periods and remissions. However, very few people make a full recovery from the disorder and a large majority of patients are unable to live independently or maintain employment for any significant period after the onset of the disorder (Hales et al, 2008). Individuals living with schizophrenia have a higher risk of substance abuse, particularly nicotine dependence, with as much as 90 percent of patients dependent on nicotine. They also use a greater amount of drugs, mostly cannabis and cocaine (Kaplan et al, 1994). In addition, schizophrenic patients tend to have a slightly lower life expectancy than the general population, with approximately 10 percent of sufferers committing suicide (Sadock & Sadock, 2004). Due to the fact that schizophrenia begins at a relatively young age and requires ongoing care for those affected, the financial cost of the disorder has been estimated to exceed the cost of all cancers combined in the United States of America, with the

overall cost estimated at $62.7 billion in 2002 (Wu et al, 2005). Although such information is not readily available for the South African population, the financial cost of the disorder is likely to be just as demanding.

Schizophrenia and the South African context

Approximately one percent of the South African population is affected by schizophrenia (Trump & Hugo, 2006). It is a pervasive disorder, causing long-lasting, significant impairments in individuals diagnosed with it. In addition to the impairments resulting from the disorder, individuals living with schizophrenia face many social problems, including stigmatisation and discrimination, both in their personal lives and in the workplace. Some employers will not hire people with a history of mental illness, while other employers dismiss or demote individuals who have been diagnosed with a mental illness, which is true for schizophrenia as well as other mental illnesses.

Trump and Hugo (2006) found that individuals living in South Africa were not only ignorant about mental illness but actually avoided seeking treatment for their symptoms as a result of their fear of being stigmatised. Hugo, Boshoff, Traut, Zungu-Dirwayi and Stein (2003) found that in South Africa, mental illness is perceived as a condition relating to stress or a lack of willpower on the part of the sufferer, instead of being seen as a medical disorder. This indicates the stigma and ignorance that is attached to mental illness (including schizophrenia), which makes it difficult for sufferers to seek treatment. It must be kept in mind, however, that in many cultures, especially in South Africa, hallucinations are sometimes viewed as a spiritual or religious phenomenon and are therefore not seen as a sign of mental illness.

In addition to this, schizophrenia makes a high demand on healthcare systems, requires ongoing care, rehabilitation and support services (Sadock & Sadock, 2004). Although a number of approaches are used when treating the disorder, most approaches typically include antipsychotic medications and therapy, whether this is provided within an institution or on an outpatient basis. Antipsychotic medications ease the symptoms of the disorder, while therapy attempts to teach affected individuals to adapt to their lifestyles and function as effectively as possible (Kaplan et al, 1994). Therefore, treatment is provided on a long-term basis, which proves to be very costly.

Many people living in South Africa do not have private medical insurance and depend on services provided by public hospitals, which can be inefficient at times. Those individuals living with schizophrenia that do have medical insurance have limited benefits for conditions related to mental illness, in that they are limited in the claims that they can make on medication and hospitalisation (Trump & Hugo, 2006). In this way, the treatment required by individuals living with schizophrenia becomes very costly for their families as well as for the government in the form of disability grants and subsidies (Hugo et al, 2003). This is a particular problem in South Africa as poverty is rife and there is a lack of resources, especially within area of public health. This is due to the fact that there are not enough hospitals, especially in the rural areas, as well as a shortage of qualified health professionals working in the area. It is for this reason that research into the early identification or prevention of schizophrenia is important and will contribute to conserving scarce resources, as well as minimising the suffering that individuals living with schizophrenia and their families experience.

Aetiology

The exact aetiological process(es) that create schizophrenia are not yet known (Sadock & Sadock, 2004). There is a broad range of possible aetiologies with a combination of factors likely causing the disorder (Brixey, Gallagher, McFalls & Parmelee, 1993). The range of possible aetiologies has been grouped into genetic influences, environmental influences and neurobiological influences (Kaplan et al, 1994).

Schizophrenia has a strong genetic element with the degree of risk being proportional to the degree of shared genes (Hales et al, 2008). However, genes alone do not account for the disorder as environmental factors play a significant role in its onset. Biological influences in schizophrenia include obstetrical complications (such as diabetes and preeclampsia), abnormal foetal development and growth (such as low birth weight), and complications during delivery (such as asphyxia) (Sadock & Sadock, 2004). The Dutch Famine study found a twofold increased risk for schizophrenia among a group of individuals born during winter from 1944 to 1946, a period of severe malnutrition for many citizens in the western Netherlands (Susser et al, 1996). In addition, Brown et al (2004) found that influenza infection in the first trimester of pregnancy increased the risk of developing schizophrenia sevenfold.

Psychosocial influences also play a role in schizophrenia, and include life stressors or adverse life events, such as the death of a loved one or a natural disaster, which could combine with an individual's predisposition to cause the disorder (Cutting, 1985). Another aspect of psychosocial influences is that of abnormal parenting. Fromm-Reichmann (1948) used the term 'schizophrenogenic mother' to describe a mother that alienates her child by her rejecting him or her. In addition, a child raised by neurotic or chronically unstable parents or parents that are chronically hostile towards one another

may have an increased risk of schizophrenia as a result of the conflicting messages received from his or her parents, termed double-bind (Bateson, Jackson, Haley & Weakland, 1956).

Neurobiological influences have also been implicated in the onset of schizophrenia and include the dopamine hypothesis, which posits that the symptoms of schizophrenia are by-products of a dysfunction in dopamine neurotransmission; however, this hypothesis does not explain the negative symptoms of the disorder (Hales et al, 2008). Neuroanatomical abnormalities have also been discovered in people living with schizophrenia. One such abnormality concerns the abnormally large lateral ventricles containing cerebrospinal fluid, which have been found in the brains of schizophrenic patients, as compared to healthy control subjects (Hales et al, 2008). In addition, individuals with schizophrenia have been found to have relatively less brain tissue. However, it is not clear as to whether this is a result of a failure to develop or a loss of tissue after development, termed cellular atrophy (Sadock & Sadock, 2004).

Thus it is clear that schizophrenia may have numerous aetiologies, spanning genetic, environmental and neurobiological areas of development. Although no consensus exists about the exact cause of the disorder, definite differences and abnormalities can be seen in schizophrenic individuals as compared to people without the disorder, evidenced in a large body of research detailing such abnormality. Much of this research has been conducted on sensory, attention-, affective- and memory-related abnormalities in schizophrenia, which can be group together under the heading of 'functional abnormalities' (Sadock & Sadock, 2004). Individuals with schizophrenia have been found to exhibit impairments in attention, information processing and learning and memory (Hales et al, 2008). They also exhibit impairments in sensory

processing, such as visual and auditory processing (Sadock & Sadock, 2004). Knowledge of these impairments is important in the study, diagnosis and treatment of schizophrenia as they may be considered to be vulnerability markers, which may be used to define schizophrenia phenotypes. In addition, these impairments may be useful in the early detection of schizophrenia, as well as the identification of individuals with a high risk of developing the disorder, which in turn could lead to early treatment and a better prognosis for the individuals concerned (Sadock & Sadock, 2004). They may also provide insight into the aetiology of schizophrenia, thus indicating the importance of such research. A more detailed account of the studies conducted in this area will be provided in the following section.

Functional abnormalities

As mentioned previously, a lot of the research conducted on schizophrenia has focussed on functional abnormalities present in individuals affected by the disorder. These abnormalities have been studied in a number of different ways, including with the use of behavioural measures as well as more technical measures such as the electroencephalograph (EEG), functional magnetic resonance imaging (fMRI) and positron emission tomography (PET). Although a great amount of research has been documented on such studies, the studies examining abnormalities in sensation and perception (and somatosensory processing in particular) will be focussed on, as these are the most relevant to this particular study.

Technical measures

fMRI studies

Numerous studies have been conducted with schizophrenic patients using Functional Magnetic Resonance Imaging (fMRI), examining a number of functional

abnormalities. One such study, conducted by Kiehl and Liddle (2001), compared the fMRI results of schizophrenic patients to healthy controls from an auditory oddball task. This task consisted of non-target, target and non-repeating random tones presented to the participants using an MRI compatible auditory sound system with noise-attenuating headphones. Participants were asked to respond as quickly and accurately as possible when they heard the target tone and not the other two tones. A significant difference was found between the schizophrenic patients and healthy controls, with the patients exhibiting significantly smaller and less extensive activation in various areas of the brain, including the anterior superior temporal gyrus and thalamus, than the healthy controls, as indicated by the imaging data. Kiehl and Liddle (2001) therefore argue that temporal lobe abnormalities are present in schizophrenia, which may account for some auditory processing abnormalities.

In another study Wible et al (2001) examined whether the functional abnormalities present in schizophrenia occur at lower levels of auditory processing. They did this by presenting a series of standard tones (the switching noises from the fMRI scanner) with a number of deviant tones embedded within them, called mismatch stimuli, to a group of schizophrenic patients and mentally healthy comparison subjects, while recording fMRI data. They found that the schizophrenic patients exhibited less activation in the superior temporal gyrus than the comparison subjects and concluded that early auditory processing is abnormal in chronic schizophrenia (Wible et al, 2001). A similar finding was noted by Kircher et al (2004), who also presented the switching noises from the fMRI scanner to a group of schizophrenic patients and healthy controls, while recording the imaging data. They noted that the patients exhibited decreased responses as compared to the controls and also failed to show a lateralisation effect, thus exhibiting dysfunctional lateralisation (Kircher et al, 2004).

In their study using fMRI, Braus, Weber-Fahr, Tost, Ruf and Henn (2002) simultaneously presented a visual stimulus (a moving 6-Hz checkerboard image projected onto a screen) and auditory stimulus (drumbeats played through headphones) to a group of schizophrenic patients and healthy controls. The fMRI data showed that the schizophrenic patients exhibited reduced activation in the right thalamus and prefrontal cortex, as well as in the left acoustic cortices of the superior temporal lobe in comparison to the healthy controls, thus indicating abnormalities in the early stages of information processing (Braus et al, 2002). Although more studies have been conducted within this area of research, the four studies mentioned above illustrate functional abnormalities in schizophrenia, as shown by fMRI data. These abnormalities are further illustrated by studies utilising EEG data, as discussed below.

EEG studies

Many studies have also been conducted with schizophrenic individuals using EEG machines to measure sensory gating, which can be described as the mechanism in the brain that blocks the access of sensory input from higher cortical areas (Boutros, Belger, Campbell, D'Souza & Krystal, 1999). In other words, it is the ability of the brain to adapt its sensitivity to incoming stimuli, and helps the brain to block out irrelevant external sensory input such as background noise. It is a non-conscious mechanism that filters out irrelevant external sensory input before it reaches the conscious awareness of the person (Jin et al, 1998). Sensory gating is measured using evoked potentials, which are the brain waves that follow sensory stimulation, recorded from the scalp (Boutros et al, 1999). They allow researchers to study people's responses to sensory inputs, for example sound, termed auditory evoked potentials. They are measured in the form of computerised averages of the brain's electrical response to stimulation, such as sound (Freedman et al, 1987).

One form of auditory evoked potential measure is the P50, where the subject is exposed to two click sounds (S1 and S2) that last 50 milliseconds each and are presented 500 milliseconds apart (Clementz, Geyer & Braff, 1998). P50 evoked potentials have commonly been used in studies examining sensory gating in schizophrenia. Mentally healthy individuals have been found to suppress the second of two clicks (S2) at an average rate of 60% to 80%, while schizophrenic individuals show a suppression of the second click at an average rate of 20% to 50% (Clementz et al, 1998). In other words, mentally healthy individuals block out the second click 60% to 80% of the time, while schizophrenic individuals only block it out 20% to 50% of the time. This finding has been replicated by numerous researchers examining sensory gating in schizophrenia (Siegel, Waldo, Mizner, Adler & Freedman, 1984; Boutros, Zouridakis & Overall, 1991; Yee, Nuechterlein, Morris & White, 1998; Hsieh et al, 2004). A smaller response to the second click (S2) indicates 'gating-out' of irrelevant auditory input, which is present in mentally healthy individuals but not schizophrenic individuals (Clementz et al, 1998). The 'gating-out' of the second click by mentally healthy individuals can be explained by the active inhibitory physiological theory, which says that when exposed to two identical click sounds, as in the P50 measure, the second stimulus (S2 in this example), because it is identical to the first stimulus (S1), presents no new information and is therefore gated out so as not to proceed to higher cortical areas for processing (Boutros et al, 1999). Thus, individuals with schizophrenia experience difficulty inhibiting irrelevant incoming stimuli in the pre-attentive phase of information processing and are therefore said to exhibit sensory gating deficits (Boutros et al, 1999).

Schizophrenic patients have also been found to exhibit visual processing abnormalities. For example, O'Donnell et al (1996) found that schizophrenic patients exhibit deficits in the perception and representation of motion, spatial relationships and orientation. Studies have also examined visual processing in schizophrenic patients, using visual evoked potentials. For example, Krishnan et al (2005) examined visual evoked potentials in schizophrenic and mentally healthy individuals and found that the individuals with schizophrenia exhibited deficits in visual processing, as indicated by their reduced power at higher stimulating frequencies. In addition, Butler and Javitt (2005) note that schizophrenic patients exhibit decreased evoked potential amplitudes, as compared to healthy controls, when completing visual tasks, thus indicating early visual cortical dysfunction. This is supported by Johnson, Lowery, Kohler and Turetsky's (2005) study, which examined temporal and spatial characteristics of hierarchical visual stimulus processing abnormalities in schizophrenic patients, and found that they exhibit a global visual processing deficit, which occurs at a relatively early stage of visual processing.

PET studies

Studies have also been conducted with schizophrenic individuals in this area using positron emission tomography (PET) scans. For example, Blackwood et al (1994), using PET, found abnormalities in smooth pursuit eye movement (the ability to track a predictably moving object with ones eyes) in schizophrenic patients, and concluded that it is associated with bilateral frontal lobe disturbance. In another study using PET, Taylor, Tandon and Koeppe (1997) compared the activation response in schizophrenic patients and healthy controls when exposed to lights flashing at four different rates. They found that the schizophrenic patients exhibited a greater degree of activation than

the control subjects and argued that further research would be needed to clarify and explain this result.

Thus, from the abovementioned studies, one can conclude that although further research is required to gain a more comprehensive understanding of these abnormalities, functional abnormalities are a feature of schizophrenia and can be examined at a basic, neuronal level of functioning. These studies are valuable in terms of understanding the possible causes of the disorder and while useful, do not provide a sufficient explanatory account for disturbances of sensory processing that occur at higher levels of processing, involving the concerted activity of various areas of the brain, and that are affected by cognitive processes such as attention and not directly measured by these methods (Luria, 1976). Therefore, these measures may not be able to provide a fully comprehensive picture of sensory processing abnormalities and are certainly not exhaustive in examining all aspects of higher levels of sensory processing. It is for this reason that empirically observable behavioural measures of sensory processing may be useful in shedding light on the behavioural manifestations of these abnormalities.

Behavioural measures

Behavioural measures are varied and involve the study of different areas of functioning, as they are sensitive to a variety of higher cortical processes, which influence observable behaviour. Studies conducted within this area of research have examined aspects such as pain processing, proprioception and smooth pursuit eye tracking in schizophrenic patients as compared to mentally healthy individuals. A description and summary will be provided for the studies conducted in each of these areas.

Pain processing

A high tolerance for pain has been observed in schizophrenic patients from as far back as the early 1900's where Kraepelin observed that schizophrenic patients tended to have a high tolerance for physical discomfort (Singh, Giles & Nasrallah, 2006). Pain insensitivity in schizophrenia has been documented in case studies, which include reports about schizophrenic patients having ailments such as a ruptured appendix, fractured bones and a perforated bowel and yet experiencing no pain, which in turn led to late diagnosis (Apter, 1981; Fishbain, 1982; Rosenthal, Porter & Coffey, 1990). Some researchers have discovered the opposite, for example, Varsamis and Adamson (1976), who note that 48% of the schizophrenic patients that they observed had complaints about pain and it was only the patients that were markedly withdrawn that did not complain about pain. However, many studies have been conducted measuring pain thresholds in schizophrenia, which point to pain processing abnormalities (Singh et al, 2006).

In one such study, Kudoh, Ishihara and Matsuki (2000) compared a group of schizophrenic patients to a group of mentally healthy controls while measuring their responses to electric stimuli. They found that the schizophrenic patients exhibited a higher pain threshold than the controls. Another study, conducted by Blumensohn, Ringler and Eli (2002) found that schizophrenic patients tend to have an increased pain threshold and pain tolerance, as compared to controls. This finding was also noted by Hooley and Delgado (2001), who found an increased pain threshold and tolerance in the relatives of schizophrenic patients. In addition, Dworkin (1994) found that schizophrenic individuals have disturbed pain-processing tendencies, which have important implications for physical health.

Proprioception

Another area of research using behavioural methods is that of proprioception, which is the sensory process guiding body position and balance (Chang & Lenzenweger, 2005). Rado (1960) argued that individuals with a genetic risk of schizophrenia exhibit deficits in proprioception, whether they actually go on to develop the disorder or not. One way in which to measure proprioeption involves measuring ones ability to judge and discriminate between various lifted weights. In one such study, Sonder (1955) found that schizophrenic individuals tend to have a decreased ability to judge lifted weights. This finding was echoed by Ritzler and Rosenbaum (1974) who found that schizophrenic individuals have elevated weight discrimination thresholds, as compared to control subjects. They concluded that schizophrenic individuals have a proprioceptive deficit, which they argued was due to inadequate sensory input (Ritzler & Rosenbaum, 1974). Further, Ritzler (1977) found evidence for a subtle proprioceptive deficit in schizophrenic patients, as compared to healthy controls, using a weight-discrimination task.

Smooth pursuit eye tracking

As mentioned previously, smooth pursuit eye tracking has been utilised as a behavioural measure in the study of schizophrenia, with studies being conducted as early as 1908. Diefendorf and Dodge (1908) (as cited in Freedman, Ross & Adler, 1998) noted that schizophrenic patients have difficulty in maintaining their visual gaze on a predictably moving target, more recently termed smooth pursuit eye tracking. This was also found by Holzman et al (1974), who noted that schizophrenic patients exhibit deviant eye tracking. They also found this true for clinically unaffected relatives of the schizophrenic patients and argued that the eye tracking dysfunction may represent a genetic marker for the disorder. This finding was echoed by Bartfai, Levander, Nybäck, Berggren and Schalling (1985), who found impairments in the

smoother pursuit eye tracking of 18 schizophrenic patients. This finding has also been replicated by numerous other researchers (Iacono, Moreau, Beiser, Fleming & Lin, 1992; Levy, Holzman, Matthysse & Mendell, 1994; Sereno & Holzman, 1995).

Although many behavioural studies examining somatosensory processing in schizophrenia have been conducted, the studies mentioned above provide evidence for sensory abnormalities present in individuals with the disorder. Behavioural measures of sensory abnormalities in schizophrenia provide insight into the construct validity of studies in this area in that the findings can hopefully, one day be 'mapped onto' anatomical findings from more technical studies examining the brain. Therefore, behavioural measures provide a different view of functioning and are useful in the study of schizophrenia. One behavioural measure in particular, and which has been used extensively with the schizophrenic population, has provided insight on stimulus control in schizophrenia. This measure is called the Kinaesthetic Figural Aftereffects task (KFA) and will be discussed in the following section.

Kinaesthetic Figural Aftereffects task

The KFA has been used in studies dating back to the 1950's and it has been argued that this instrument can assist researchers to define people's perceptual styles. Here, a brief history on the instrument will be provided, followed by a description of how it works, after which a summary of the studies using the KFA will be provided.

The KFA was originally based on the work of Petrie, Collins and Solomon (1958) who were investigating the area of relative pain thresholds as evidencing nervous systems of differing levels of sensitivity. It has since been used to classify perceptual styles in

psychiatric and normal populations, specifically focussing on stimulus control in schizophrenia (Kuster, Harrow & Tucker, 1975).

The actual procedure of the task, as described by Petrie, Holland and Wolk (1963), requires the subject to hold a rectangular wooden bar in the one hand (the issue of handedness is a debated one in this procedure) and a tapered wooden bar in the other, which consists of various gradations. The subject then moves his or her thumb and forefinger along the two bars until they feel equal in width and therefore makes an estimation of the size of the standard block on the tapered block. This procedure is repeated four times. The subject is then given another wooden block, which is wider than the standard and tapered block and is told to rub this block with his or her thumb and forefinger for a period of 30 seconds (this is called interpolation). The subject then makes more estimations as was done previously, on the tapered bar. This rubbing is believed to induce satiation if it is to occur, which Köhler (as cited in Petrie et al, 1963) described as a phenomenon where perceptual intensity decreases after prolonged stimulation with a stronger stimulus. Therefore, the KFA presents a fixed intensity stimulus (size), followed by repeated kinaesthetic stimulation of a different intensity (rubbing). When the original stimulus (size) is presented again, it will either appear less intense or smaller, unchanged or more intense or larger (Kuster et al, 1975).

The KFA purports to examine the way in which people respond to sensory input on repetitive trials of psychophysical estimation. That is, it measures responses to somatosensory satiation and overstimulation. It can also be utilised as a measure of the effects of sensory ‘overload’ on estimation but is not designed to measure estimation accuracy. According to the original theoretical paradigm in which this procedure was initially developed in the 1970s, individuals can be classified into three groups depending on their response to a sensory satiation procedure.

The first group were labelled as ‘reducers’ and included individuals who experience sensory input as less intense following a satiation procedure, as they are said to have hypersensitive nervous systems (Sales & Throop, 1972). By reducing sensory input, they are said to be able to cope with intense stimuli. It was suggested that people whose estimates dropped following a satiation procedure could be exhibiting a compensation or adaptation to a sensory overload. People whose estimates of stimulus intensity increased following satiation were dubbed ‘augmenters’ (Sales & Throop, 1972). ‘Augmenters’ were said to perceive sensory stimuli as more intense following satiation. It was argued at the time that augmenters have ‘hyposensitive’ nervous systems and tend to exaggerate or ‘blow up’ stimuli. Finally, ‘moderators’ (those whose estimates of stimulus intensity remained unchanged) experience sensory input as relatively unchanged. This perceptual modification following stimulation is believed to indicate the style of perceptual control that an individual exerts on his or her experience of the world (Kuster et al, 1975). Thus, estimation accuracy is not important with regards to the KFA task, but rather how individuals’ estimates react to somatosensory satiation and overstimulation.

A number of studies have been conducted using the KFA, beginning with the work of Petrie and her colleagues in the 1950’s and 1960’s and continuing until the late 1970’s, where the use of the electroencephalograph (EEG) overtook the use of this instrument. In one of the first studies conducted in the area, Petrie (1967) (as cited in Mishara, Baker & Parker, 1973), using the KFA, found that individuals that judged the tapered wooden block as being smaller after satiation weaken the intensity of incoming stimulation, while individuals that judged the block as being larger following satiation amplify stimulus intensity and those who judged the block as unchanged neither weaken or amplify the intensity of the stimulus. In addition, she found that individuals with schizophrenia tended to be reducers.

A few years later, Houpt, Tucker and Harrow (1972) conducted a study using the KFA with a group of 'classical' schizophrenic individuals or individuals exhibiting the typical symptoms of the disorder, a group of 'latent' schizophrenic individuals or those individuals exhibiting symptoms of the disorder but without a psychotic episode and a group of psychiatric patients with a different diagnosis, such as depression. They found that the 'classical' schizophrenic individuals exhibited significantly more reducing behaviour than the group of psychiatric patients without diagnoses of schizophrenia. The 'classical' schizophrenic individuals also exhibited significantly more reducing behaviour than the 'latent' schizophrenic individuals (Houpt et al, 1972).

In their study, Sales and Throop (1972) administered the KFA task to 35 university students and also measured their sensitivity to stimulation by exposing them to a number of tones at different frequencies. They found a strong correlation between the sensitivity of the students to auditory stimulation and their scores on the KFA in that the KFA reducers were relatively insensitive to auditory stimulation as compared to the KFA augmenters who were relatively sensitive to incoming auditory stimulation. In addition, Sales and Throop (1972) examined whether KFA scores were at all related to Pavlov's theory of 'strength of excitation of the nervous system', which states that people with 'strong' nervous systems are relatively hyposensitive to weak stimuli but process intense stimuli effectively, while people with 'weak' nervous systems are sensitive to weak stimuli and overwhelmed by intense stimuli. They argued that KFA scores do in fact appear to align with Pavlov's theory.

Ritzler and Ebner (1973) utilised the KFA task in their study comparing a group of college students, a group of individuals with acute schizophrenia and a group of individuals with chronic schizophrenia. They found the group of college students to be augmenters as compared to the group of chronic schizophrenic individuals, who were reducers. The group of acute schizophrenic individuals were also reducers on the KFA

task, but not to the same degree as the chronically schizophrenic individuals (Ritzler & Ebner, 1973). In another study, Kuster et al (1975) administered the KFA task to a group of classical schizophrenic patients, latent schizophrenic patients (as defined above) and a group of non-schizophrenic psychiatric inpatients. They found that although the group of schizophrenic patients tended to reduce the intensity of incoming stimuli in the KFA task, no significant difference was found between the three groups of patients.

Lastly, Schooler, Buchsbaum and Carpenter, (1976) administered the KFA task and an EEG technique of average evoked response to a sample of both acute and chronic schizophrenic individuals. They found that individuals classified as augmenters on the KFA task were also classified as augmenters on the EEG average evoked response task and vice versa. They concluded that a consistent dimension of stimulus intensity control does exist. Thus, it has been argued that individuals with schizophrenia tend to be hypersensitive to external stimuli (Jin et al, 1998). Reducers are said to have a compensatory mechanism in order to prevent or avoid sensory overload due to the fact that they have hypersensitive nervous systems. That is, they reduce or 'damp down' sensory input, which results in them perceiving stimuli at a decreased intensity (Buchsbaum & Silverman, 1968). No findings regarding estimation accuracy were reported in the abovementioned studies. In addition, findings of relative accuracy between schizophrenic patients and individuals without the disorder on the KFA task have not consistently been reported in the published studies.

While some debate exists over the reliability of the procedure (with arguments made on both sides), serious questions can and have been posed around the construct validity of what precisely is measured by the KFA. A variety of arguments exist suggesting that what is being measured are variances in processes such as attention, memory or a

complex interaction of all of these. As such the older theoretical paradigm of 'augmenting, reducing or moderating' nervous systems may lack sufficient empirical support (Schooler et al, 1976). Whether or not credence is given to the paradigm of augmenting-reducing, the instrument does appear to have some face validity in measuring capacity for higher-order cortical suppression (or enhancement) of sensory stimuli, such as that proposed to be at work in the 'sensory gating' paradigm, or more recently, in that of the selective-filter paradigm of research in schizophrenia (Schooler et al, 1976). These paradigms will be discussed in more detail next.

Explanatory paradigms for somatosensory abnormalities

As mentioned previously, a number of paradigms have been utilised to explain the findings of studies using the KFA. The first of these paradigms consists of genetic-based theories, which argue that the somatosensory abnormalities observed in schizophrenic patients using the KFA can be explained by their genetic makeup. Another paradigm attempting to explain the findings from studies using the KFA consists of sensory gating theories. Here, it has been argued that the mechanism for sensory gating is faulty in schizophrenic individuals. It has also been argued that as a result of sensory gating failures, schizophrenic patients tend to become overwhelmed by sensory inputs and compensate for this by suppressing their response to these inputs. Another idea that has been put forward is that which argues that schizophrenic patients fail to integrate information at a higher cortical level and thus fail to react appropriately to incoming stimuli. Although no one theory has been completely proved or disproved, all of the abovementioned theories provide possible explanations for the somatosensory processing abnormalities present in schizophrenic patients and will therefore be discussed in more detail below.

Genetic-based theories

Although no consensus exists about the aetiology of schizophrenia, it has a strong genetic element with the degree of risk proportional to the degree of shared genes (Hales et al, 2008). This is indicated by studies that have been conducted with monozygotic (identical) twins, who share identical genes, that have found a 50 percent chance that both individuals will develop the disorder. That is, if one twin has schizophrenia, the identical sibling has a 50 percent chance of developing the disorder. Dizygotic twins have a lower risk, as they do not share genes and thus a non-identical twin sibling of an individual with schizophrenia has the same risk of developing the disorder as any other first-degree biological relative of that individual (Sadock & Sadock, 2004). Studies focused within the realm of adoption have found that there is a higher risk for schizophrenia among the children of mothers with the disorder, even though the children are raised apart from her. Therefore, there is strong evidence implicating the role of genes in schizophrenia. However, no one gene is responsible for the disorder but rather a complex combination of genes, which create vulnerability for the disorder. Interestingly, many genes that have been linked to schizophrenia are related to neurodevelopmental processes, which assist in establishing neural networks, such as synapse formation and neuronal migration (Hales et al, 2008).

One theory argues that the genetic characteristics associated with schizophrenia may cause the somatosensory abnormalities mentioned earlier, whether they cause it directly or indirectly. Meehl (1962) argued that all individuals that go on to develop schizophrenia have a basic propensity (a genotype) for the disorder, which he termed schizotypy. He argued that individuals with schizotypy (or schizotypes) that do not go on to develop schizophrenia still exhibit subtle neurocognitive symptoms, indicating schizotypy. Therefore, Meehl's concept of schizotypy represents a vulnerability to

schizophrenia that certain individuals have, which is indicated by their neurocognitive symptoms. He argued that a "spatial-kinesthetic-vestibular dysfunction", in other words a proprioceptive deficit, is a characteristic of schizotypy and as a result, schizotypes are likely to exhibit some abnormality in this area of somatosensory processing (Chang & Lenzenweger, 2005).

In order to study schizotypy, one could compare the direct biological family members of schizophrenic individuals with healthy controls in tasks involving somatosensory processing. Clementz et al (1998) did just this in their study of clinically unaffected first-degree biological relatives of schizophrenic individuals, which examined the P50 response to paired-click sounds. They found that the relatives of the schizophrenic patients had worse P50 suppression than normal subjects with no schizophrenic relatives. However, the schizophrenic patients themselves still exhibited the worst P50 suppression. In a similar study, Siegel et al (1984) found that half of the first-degree biological relatives of the schizophrenic patients tested using the P50, exhibited sensory gating deficits. Chang and Lenzenweger (2001) found that first-degree biological relatives of schizophrenic individuals exhibit decreased touch sensitivity and thus exhibit impairments in touch processing. They also found that the relatives of schizophrenic patients performed worse on a graphesthesia task, which involves complex somatosensory processing, than a group of controls (Chang & Lenzenweger, 2004). In addition, Hooley and Delgado (2001) found that relatives of schizophrenic patients tend to have increased pain thresholds.

Another way in which to study schizotypy would involve the linkage of a specific, physiological phenotype to a genetic locus, termed linkage studies (Freedman et al, 1998). Linkage studies provide insight into schizophrenia as they confirm the fact that

physiological abnormalities found in schizophrenic patients and their relatives, are related to neurobiological abnormalities, which in turn are related to genes. When a positive linkage is made, it indicates that a physiological phenotype is closely related to a neuronal defect caused by a genetic abnormality (Freedman et al, 1998). The fact that first-degree biological relatives of schizophrenic patients exhibit physiological abnormalities, as mentioned earlier, adds credence to this area of study.

Although progress has been made in the abovementioned areas of study, a lot of uncertainty still exists around how somatosensory abnormalities exhibited by schizophrenic individuals and their relatives, link to specific genes and to clinical outcomes more generally (Freedman et al, 1998). Thus, many questions still exist about all sensory abnormalities and their relevance to the study of schizophrenia.

Sensory gating theories

Another approach to understanding somatosensory processing abnormalities in schizophrenia is not focused on genetic studies but instead attempts to provide an explanation couched in functional terms. In this area of study, researchers have tried to identify underlying constructs in the form of mechanisms of sensory processing, which, when they are not working normally, may lead to a number of the observed sensory abnormalities. One such construct that has historically received much attention is that of sensory gating. As mentioned previously, sensory gating is the mechanism in the brain that blocks the access of sensory input from higher cortical areas and is the brain's ability to adapt its sensitivity to incoming stimuli (Boutros et al, 1999).

Within this area of research, there are two overall explanatory positions, namely that schizophrenic patients experience sensory gating difficulties and as a result suppress

sensory input as they tend to be overwhelmed by it and/or that schizophrenic patients fail to integrate sensory input at higher cortical levels. The first explanatory position consists of researchers who have argued that sensory gating is faulty in schizophrenic patients due to genetic factors. They argue that the actual sensory gating mechanism in the brain has something to do with the way in which somatosensory information is sensed and processed. Gulli and Rosick (2005) argue that individuals with schizophrenia are unable to filter sensory stimuli and therefore tend to have enhanced perceptions of sounds, colours and other features of the environment, while Freedman et al (1987) argue that schizophrenic individuals are unable to filter out noise from meaningful inputs due to genetic deficits in sensory functioning and possible dopamine-metabolism abnormalities (Freedman et al, 1987). Baker et al (1987) argue that deficient sensory gating is a characteristic of schizophrenia, while Jin et al (1997) noted that schizophrenic individuals often refer to their decreased ability to focus on external sensory stimuli and their excessive awareness of background noises. These studies point towards the abnormal sensory gating present in schizophrenic individuals. Sensory gating has been measured using the P50 measure mentioned earlier. The finding, as mentioned previously, that schizophrenic patients gate out the second click to a much lesser degree indicates that they tend to have impaired auditory gating (Siegel et al, 1984; Clementz & Blumenfeld, 2001; Hajos, 2006).

Related to the study of sensory gating deficits in schizophrenia is the area of study, which focuses on the apparent neurological sensitivity of schizophrenic patients to stimuli. Howe (1991) noted that individuals with schizophrenia are unable to filter out meaningless, background noise in the way that people without the disorder do, causing their hearing to become extremely sensitive and making ordinary sounds seem high-pitched and even deafening. Thus, people with schizophrenia may be able to hear

sounds that would normally go unnoticed. Howe (1991) also noted that their other senses are also be very sensitive, such as their vision, leading them to experience very dull or very bright colours in their environment.

Kaplan et al (1994) therefore argue that schizophrenic individuals are unusually sensitive to sensory stimuli from the environment. There are numerous theories regarding why individuals with schizophrenia are excessively sensitive to sensory stimuli in the environment. One such theory, supported by substantial evidence, argues that sensory gating failures lead to sensory overload in schizophrenic individuals (Judd, McAdams, Budnick & Braff, 1992), while many other studies have found that individuals with schizophrenia have impaired sensory gating mechanisms, which leads them to experience an overload of sensory information at a conscious level (Patterson et al, 2003).

Another general characteristic of schizophrenia, indicated by studies conducted using the KFA task, is the apparently contradictory finding that schizophrenic patients exhibit a reduced response to stimuli. That is, schizophrenic patients have been found to be 'reducers' on the KFA task as they experience sensory input as less intense. These two contradictory findings have been integrated into a fairly common understanding of schizophrenia; namely that because schizophrenic patients experience sensory overload due to sensory gating deficits, they compensate for this by 'dampening down' sensory input, which results in them perceiving stimuli at a decreased intensity (Buchsbaum & Silverman, 1968). That is, due to the fact that schizophrenic individuals tend to be hypersensitive to sensory input and have sensory gating deficits, they may possess a mechanism that decreases the intensity of a stimulus before it is processed and reaches their conscious awareness.

The second trend evident in sensory gating studies involves the notion that schizophrenic individuals exhibit sensory gating abnormalities because of a failure to integrate the information at a higher level in the brain, which inhibits their ability to react appropriately. That is, schizophrenic individuals experience a failure in functional connectivity, which limits cortical integration and network activation, which is needed for somatosensory processing tasks (Peled et al, 2001). Imaging studies have provided evidence indicating that schizophrenic individuals experience disturbances in their functional cortical integration, and thus that different cortical regions are disconnected from one another. In their study using an EEG, Peled et al (2001) found that schizophrenic patients exhibit fronto-temporal neural network failures during a working memory task, thus indicating a disturbance in the functional connectivity between these areas. This would imply a different mechanism underlying somatosensory 'insensitivity', also operating at a hierarchically 'higher' level in the nervous system.

Thus it is clear that the findings of studies conducted on sensory gating in schizophrenia are contradictory with one set of findings providing evidence for the suppression of stimuli, while the other set of findings provides evidence for a lack of integration at higher levels of processing in the brain. It is for this reason that a lot more research is needed in this area, both at a more technical level, as well as at a behavioural level, to explore the mechanism responsible for processing abnormalities in schizophrenia, whether it is genetically based or due to the disorder itself. It is here that the KFA task, discussed earlier, may have potential in that it does not just measure accuracy, but it also looks at sensation-change over time and can therefore answer some of these questions. Because the KFA task measures changes in processing, it may provide insight into the debate between sensory gating versus failure of sensory

integration in high cortical areas. However, the original theoretical paradigm used to explain the findings, namely augmenting and reducing, is outdated. Therefore, it may make sense to reinterpret the previous findings using the KFA task within the sensory gating paradigm, rather than the paradigm of augmenting and reducing.

However, before this can be done, measurement issues surrounding the KFA task need to be addressed. One of the main concerns is about the reliability of the KFA task. Some researchers have found it to be a reliable measure while others have not (Sales & Throop, 1972). Schooler et al (1976) found the KFA task to be a reliable procedure on the basis that they replicated earlier findings of studies using the instrument. In addition, they discovered that KFA augmenters and reducers are also Average Evoked Response (AER) augmenters and reducers, as measured using an EEG, thus indicating the differences between the two groups at a basic, neuronal level.

Although a few studies attest to the reliability of the KFA task, more research needs to be done to establish its reliability and whether studies using the KFA can be replicated. That is, it needs to be established whether one can reliably discriminate between schizophrenic individuals and healthy controls on the basis of the results obtained from the KFA task. However, reliable discrimination between the two groups could prove to be difficult due to the attentional deficits exhibited by schizophrenic patients as the length of the task may prove laborious, difficult and may, as a result, have an impact on the findings. In order to address this, a shortened version of the original KFA task could be utilised with schizophrenic populations. However, the reliability of a shorter KFA task would have to be established to determine whether the same discriminations could be made as those made using the longer version of the KFA, as well as whether the previous results could be replicated, hence the reason for this particular study.

Research questions

Does a shortened version of the Kinaesthetic Figural Aftereffects task demonstrate the same internal consistency as the original version of the instrument, when comparing a schizophrenic versus a control population?

Do individuals with schizophrenia show significantly different variations in sensory sensitisation following satiation in comparison to healthy, matched controls on a shortened version and a self-adapted version of the Kinaesthetic Figural Aftereffects task?

Can a repeated measures component to the design provide further evidence for test-retest reliability for the shortened KFA procedure?

Hypotheses

Question 1

With regards to the first research question, it is hypothesised that a shortened version of the Kinaesthetic Figural Aftereffects task will yield the same internal consistency as the original version of the instrument, when comparing a schizophrenic population with a control population.

Question 2

On the basis of previous research, it is hypothesised that a significant difference will be found between the schizophrenic and control groups on the shortened version of the Kinaesthetic Figural Aftereffects task, using a chi square analysis. More specifically, it is hypothesised, using a chi square analysis, that the control group will demonstrate a significant increase in the post-interpolation measure compared to the group of

schizophrenic patients, while the group of schizophrenic patients will demonstrate a significant decrease in the post-interpolation measure as compared to the control group.

Question 3

With regard to the third and final research question, it is hypothesised that a repeated measures component to the design of the Kinaesthetic Figural Aftereffects task will provide further evidence for test-retest reliability for the shortened version of the KFA task.

Chapter two
Methods

This chapter will discuss the methodological procedure followed in conducting this research and collecting the data needed to answer the research questions. The discussion will include the research design, the sample, the procedure including ethical considerations, the instruments used, as well as the statistical analyses performed on the data.

Research Design

The nature of the data collected for this study in addressing the research question, namely whether a shorter version of the KFA task yields the same capacity to discriminate, as the original task when used with a schizophrenic and normal sample, was quantitative data. The sensory sensitivity of the participants following interpolation was also examined to establish whether different variations occurred within the same individual over numerous trials (within subjects), as well as whether different variations occurred between the two groups (between subjects). Related to this was the collection of quantitative data to establish whether a self-developed repeated measures component in the design would yield further evidence for reliability for the KFA task, as well as the further clarification of the nature of the construct under examination. The design of this study is therefore a non-experimental, quantitative research design.

Sample

The sample in this study consisted of 32 individuals living with schizophrenia and 32 individuals with no diagnosis of mental illness and with no first-degree biological relatives diagnosed with schizophrenia, who are described in this report as ‘controls’.

The diagnosis of schizophrenia was established in this study as being the most current diagnosis on file of the organization through which participants were sampled, together with the opinion of the head of each organization that schizophrenia was indeed the correct diagnosis. Many participants confirmed this diagnosis themselves in conversation.

The sample of individuals living with schizophrenia was drawn from three separate organizations. The sixteen individuals living with schizophrenia who participated in the initial study were included from Gateway House, a community based care provider for sufferers of psychiatric illness, from the previous study conducted in this area (Spyrelis, 2008). Of the sixteen schizophrenic patients who participated in the present, second leg of the study, twelve were included from Thandanani Centre. Thandanani is a non-profit organization that provides care for sufferers of psychiatric illness, primarily individuals with psychotic disorders such as schizophrenia and bipolar disorder. These two organizations provide inpatient care that is voluntary, and do not retain individuals against their will and are therefore not psychiatric institutions. Another four schizophrenic patients were sampled through the Schizophrenia and Bipolar Disorders Alliance (SABDA), a non-profit support group that provides outpatient services to individuals living with schizophrenia and bipolar disorder, as well as to their families and caregivers. Although all of the individuals living with schizophrenia that formed part of the sample for this study suffer from a chronic form of schizophrenia, none of them are in the acute stage of the disorder, are all on medication and receive some form of care tailored to the disorder.

The control or comparison sample consisted of 32 individuals with no history of mental illness and no first-degree biological relatives with schizophrenia. They were

matched on age (to within five years of age), race and gender to the group of individuals living with schizophrenia. Sixteen comparison individuals were included from the previous study conducted in this area, while the other 16 individuals were included in the current study.

Participation in this study was on a volunteer basis and the samples can therefore be classified as non-probability convenience samples. That is, due to the fact that the participants were asked to volunteer, not everybody in both populations had an equal non-zero probability of being included in the samples and thus the samples were non-probability (Huck, 2004).

Instruments

The Kinaesthetic Figural Aftereffects task (KFA) was administered to the participants. The instrument used was like that described and used by Ritzler and Ebner (1973) in their study on stimulus intensity among individuals with schizophrenia and those without the condition. It consists of four wooden blocks of different shapes and sizes, which were made specifically for this research according to the exact dimensions described here. Namely, a 1.5-inch (38.1mm) standard wooden block, a tapered, cone-shaped comparison block, with a thickness of 0.5-inches (12.7mm) at the narrow end and 2.5-inches (63.5mm) at the widest end, marked into 26 gradations, starting from the wide end with each gradation being 1/16 of an inch narrower than the one before it, and two interpolated stimulation blocks of 2.5-inches (63.5mm) each. All four wooden blocks were 10.12-inches (25.7mm) in length. These blocks were made out of pinewood and had smooth sides to prevent the participants from getting splinters. The correct estimate of the thickness of the standard wooden block on the tapered wooden

block is at gradation 13, although noting that actual estimation accuracy is not a central measure of this procedure. The KFA task requires participants to be blindfolded, however, in order to lessen the anxiety in the participants of this study, a screen covered with material was constructed and used instead of a blindfold. Participants held their hands under the screen and manipulated blocks on the other side where they could not see them.

For the administration of the KFA, participants were given the standard wooden block in their dominant hand and the tapered wooden block in their other hand and asked to find the thickness of the standard block on the tapered block, without being able to see their hands, which were placed underneath the screen. The participants made an estimate by moving their non-dominant hand along the tapered block until they reached a point which felt equal in width to the standard block in their dominant hand (Schooler et al, 1976). The participants made one estimate and then lifted their hands off the blocks for a moment and then made a second estimate. Following this, the participants rubbed the two interpolation stimulation blocks (with their hands still out of sight) for a period of about 30 seconds. They then made a third estimate using the standard and tapered blocks, as was done previously (Schooler et al, 1976).

Another instrument, identical to that of the KFA, was developed for the present study with a different sized standard block. The standard wooden block for this instrument was 1.69-inch (43mm) thick instead of 1.5-inch (38.1mm) as in the original KFA instrument. Therefore, the correct estimate of the standard wooden block on the tapered wooden block is at gradation 16 instead of gradation 13, as in the original KFA instrument. This task is not formally part of the original KFA task but was conceptualised so as to possibly offer further reliability information on the KFA, as

well as to assess whether an individual's accuracy is at all related to the width of the correct estimate. That is, whether they are more or less accurate when estimating a narrower width on the wooden blocks. The procedure for this task was identical to that discussed above.

Procedure

A verbal presentation of the study and its requirements was first made to the participants. They were told that their participation was completely voluntary and that they were free to withdraw from the study at any time if they no longer wanted to participate. They were also given a Participant Information Sheet (see Appendix C) with information about the study as well as the contact details of the researcher and supervisor. Participants were asked to sign a Participant Consent Form (see Appendix D), which stated that participation was completely voluntary, that they may refuse to do anything that they did not want to do, that they could withdraw at any time, that no identifying information would be included in the report, that their responses would be kept confidential and that the research may be published in a journal in future.

In order to prepare the schizophrenic patients for the research and to allow them to become comfortable with the researchers, a number of activities were conducted with them before the actual tests were administered. These activities were conducted with the schizophrenic patients in both this study and the previous study (Spyrelis, 2008). At all three organizations, the verbal presentation was given in the presence of the director or group facilitator of the relevant organization as well as the supervisor of this study, who has extensive experience working with the schizophrenic population.

Three activities were then conducted with the schizophrenic patients before the research was conducted. The first of these activities was one where household objects, such as candles, spoons and small bottles, were placed in a black bag (and therefore out of sight) and the residents had to put their hands into the bag, choose an object and try to guess what the object could be. The residents then had to place their hand in another black bag with identical objects to that of the first bag, in order to find the matching object without being able to see it. The screen developed for use with the KFA was then introduced by placing the screen in front of the residents and asking them to match 3 lids to 3 separate but similar bottles while keeping their hands underneath the screen and therefore out of sight. The third exercise also included the screen and the residents were asked to place their hands underneath it and attempt to tie the shoelaces of two shoes placed behind it. All three exercises were timed and were presented as a type of fun competition in order to encourage the residents to participate. The residents' scores were then announced back to the group. These kinds of activities are all fairly common in the group sessions of all three organizations.

Demographic details including age and gender were recorded for each participant in order for him or her to be matched to the control group. Each participant's handedness or dominant hand was also recorded.

The Kinaesthetic Figural Aftereffects Task

The Kinaesthetic Figural Aftereffects task was administered to all of the individuals that chose to participate in the study. As mentioned previously, the KFA requires participants to be blindfolded but a large wooden screen covered with material was used instead so as to not cause anxiety or discomfort amongst participants. Participants placed their hands underneath the screen and out of sight during the administration of

the KFA. The original KFA task was administered according to the description provided above.

Following the first administration of the KFA (the shortened version of the original KFA), the participants were then asked to look at 11 different pictures of natural scenes, including mountains and animals, and choose their favourite one or two pictures out of the set. This was done to distract the participants from the KFA task by stimulating their visual sense, so as to change their focus from their tactile sense to their visual sense. This was necessary for the task that followed in that it hopefully eliminated the sensitising effect of the satiation procedure in the first set of trials. Pictures of natural scenes were chosen for this study, as they are neutral and would not be likely to evoke strong emotions in the participants.

After looking at the pictures, the procedure for the original KFA task was repeated with the participants, except with a different sized standard block, as discussed previously. Thus, the participants once again placed their hands underneath the screen with their dominant hand on the standard wooden block and their non-dominant hand on the tapered wooden block. The participants then estimated the size of the now larger standard block on the tapered block. They then lifted their hands for a moment and made a second estimation. They then rubbed the two interpolation stimulation blocks for a period of about 30 seconds and then made a third estimate, as was done previously.

Although the following tasks were not performed in the more recent study, it is worth noting that in the previous study, two self-developed measures were administered following the first KFA task. The first measure was called the ‘sandpaper’ test and required participants to make estimations about the texture of different pieces of

sandpaper without being able to see their hands. The second measure was called the 'ball-bearing' test and required participants to match the size of different ball bearings with their hands out of sight, behind the screen (Spyrelis, 2008). These tests were developed with the aim of measuring sensory 'sensitivity' but were not found to be sufficiently reliable and were thus excluded from the present study. As mentioned previously, the second measure developed for this study but based on the original KFA was developed specifically for this study and was not used in the previous study.

All participants were given feedback on their scores at the end of the tasks and were also informed that further feedback regarding the overall study would be provided at a later stage through a verbal presentation. The participants from Gateway House have already received a verbal presentation about the results of the previous study.

Ethical considerations

All participants included in this study, specifically the individuals living with schizophrenia, were able to give their informed consent to participate in this study without requiring the signature of a guardian. Informed consent was obtained from each and every participant before the research commenced when participants signed the consent form (see Appendix D). In addition, the activities mentioned previously were conducted with the schizophrenic patients to ensure that they were comfortable with the researcher and research setting. At both SABDA and Gateway House, all residents or members were given the opportunity to participate, including those without a diagnosis of schizophrenia. This was done so as to prevent the individuals with schizophrenia feeling as though they had been singled out. However, only the data from the schizophrenic residents were used for the study. All procedures taken to

reduce potential anxiety with schizophrenic participants were carefully discussed and planned before the time with the respective head of each organisation.

In addition and as mentioned previously, a screen covered with material was constructed for the activities rather than blindfolding participants, which could have been anxiety provoking. All participants were assured that their responses would remain as confidential as possible, noting that the only people that would have access to them would be the other participants present, the researcher and the supervisor of the study. Further, it was explained that their responses would be anonymous in the final report, forming part of groups of combined scores analysed using statistical procedures. It was also explained to the participants that they could refuse to participate in the study or withdraw at any time without the threat of any negative consequences. This was explained in the verbal presentation and in the Participant Information Sheet (see Appendix C). Ethical clearance was obtained from the Human Research Ethics Committee (Medical) of the University of the Witwatersrand for the previous study and was extended to include the present study (see Appendix A and Appendix B). In addition, a permission letter was obtained from Dr Pieter Grobbelaar, the head of Gateway House (see Appendix E), Jackie Hinks, the director of Thandanani (see Appendix F) as well as Sheila Lahoud, a committee member at SABDA (see Appendix G) in order to proceed with the study.

At last follow-up with each of these people, participants had been reported as generally having enjoyed the research activities, and that no adverse effects had been observed in any of the participants of the study.

Data analysis

The analyses used to address each of the research questions will be provided below. General summary statistics were collated in order to describe the sample, while Kolmogorov-Smirnov tests were conducted to establish whether the data were normally distributed. In addition, correlations were conducted to establish whether the control measures, such as age and handedness had any effect on the performance of the participants on the KFA tasks. Possible cohort effects were explored with the use of a two independent sample t-test, as well as a Mann-Whitney U analysis. In order to address the first research question, reliability analyses were conducted, namely correlations, as well as paired samples t-tests. With regard to the second research question, a repeated measures ANOVA, chi-square test and paired samples t-test were conducted in order to establish whether the two groups exhibited significantly different variations in sensory sensitisation following satiation on both KFA tasks. A Pearson's product-moment analysis and mixed ANOVA were then conducted in order to address the third research question regarding test-retest reliability and the overall estimation patterns of the two groups on both KFA tasks.

Both the original KFA task and the self-adapted KFA task created for this study consisted of scores that are interval scale, as the possible estimates ranged from zero to 26, with equal intervals of one centimetre between them (Neale & Liebert, 1986). The correct estimate on the original KFA task was the part of the tapered wooden block marked number 13, while the correct estimate on the self-adapted KFA task was the part of the tapered wooden blocked marked number 16, although the participants' estimation accuracy was not central to the findings of this study. Raw scores were obtained from the trials on each instrument, indicating the actual number on the

wooden block, measured at intervals of .25cm (or quarters of a centimetre). The raw scores were later used in the data analysis, without being coded.

Intervening variables

Intervening variables in this study, such as handedness, age, gender and race were controlled for. Handedness was controlled for by ensuring that the participants used their dominant hand when making the estimations on the trials. As mentioned previously, age was controlled for in this study by matching both groups on age to within five years of each other. Gender and race were controlled for by matching the control subjects to the schizophrenic patients. Summary statistics were calculated for age, race, handedness and gender in order to describe the two samples. In addition, a Spearman's rho correlation was calculated for age on the original KFA task in order to determine whether age was correlated to the measure and therefore, whether it influenced the performance of the participants on the task. The same was done for the self-adapted KFA task and age, except using a Pearson's product-moment analysis as the scores met parametric assumptions.

Summary statistics

Summary statistics were calculated in order to describe the sample. This included the calculation of the mean on variables such as age, gender, race and handedness. The mean was also calculated for the scores obtained from the KFA task and the self-adapted KFA task for both groups. Distribution analyses were conducted in order to test for the normality of scores obtained from both KFA tasks. A significance level of .05 ($\alpha = .05$) was set for all statistical procedures.

Reliability

A Spearman's rho correlation was conducted using the scores from the original KFA task (as the scores were not normally distributed) in order to determine whether the scores were correlated with each other and therefore, whether they were reliable. A Pearson's product-moment correlation was conducted using the scores from the self-adapted KFA task designed for this study, as the scores were normally distributed, as shown by the Kolmogorov-Smirnov procedure. This was done in order to determine whether the scores were correlated with each other, and thus, whether they were reliable.

In order to determine the test-retest reliability of the self-adapted KFA task used in this study, a Pearson's product-moment correlation was conducted using the difference between the second and third trial for the shortened version of the KFA task and the self-adapted KFA task. In other words, this test was run to establish whether, for example, a participant that overestimated in the shortened version of the KFA task would also overestimate in the self-adapted KFA task.

Control measures

In order to establish whether the control measures had an influence on the performance of the participants on both of the KFA measures, a number of correlations were calculated. A Spearman's rho analysis was conducted using the variables of age and the scores of the participants on the original KFA, in order to establish whether age had any influence on the performance of the participants on the KFA. A Pearson's product-moment correlation was conducted using the variables of age and the scores of the participants on the self-adapted KFA for this same reason. In addition, a Spearman's rho analysis was conducted in order to determine whether gender

correlated with the scores obtained by the participants on both KFA tasks. This was done as gender is a nominal scale variable.

Cohort effects

Due to the fact that the samples were tested at different times, with the first study having been conducted approximately two years previous to this study, the data was analysed to explore whether any cohort effects were present. Cohort one consisted of the scores that had been captured in the previous study for 16 control subjects and 16 individuals with schizophrenia, while cohort two consisted of the scores captured in the present study, which consisted of a further 16 control subjects and 16 individuals with schizophrenia. A two independent sample t-test was conducted to compare the age of the two groups in both cohorts. A Mann-Whitney U test was conducted to compare the gender of the two groups in both cohorts.

Summary statistics were calculated for both KFA procedures in order to determine the mean and standard deviations for the KFA scores across the two cohorts, per group. A two independent sample t-test was then conducted to compare the two groups in terms of the scores that they had obtained for the second and third trials of the original KFA task, as per cohort. This could not be done for the self-developed KFA as it was only used for the second cohort.

T-tests

A paired samples t-test was conducted as part of the data analysis using the means of the two groups (that is, the control group and group of schizophrenic patients) on each of the three trials of the shortened version of the KFA task. This was done in order to

determine whether a significant difference exists between the two groups in terms of their estimates on each of the three trials of the KFA task.

Another paired samples t-test was conducted between the second and third trial of the shortened version of the KFA task for the entire sample, in order to determine whether a significant difference exists between the two trials.

Repeated measures Analysis of variance

A repeated measures ANOVA was conducted in the previous study conducted, producing results that were close to being significant (Spyrelis, 2008). This procedure was also conducted in the current study and was chosen due to the fact that it shows the interaction between two effects. That is, whether the group that one is in (control group versus group consisting of individuals with schizophrenia) has an effect on how one responds to interpolation (somatosensory satiation), the central research question in the previous study and this study. More specifically, the repeated measures ANOVA in this study was utilised to assess the interaction between group (control or resident) and the pattern of change in the mean scores occurring in the second and third trials of the KFA task. Only these trials were used because the first score of the original KFA task was not only not normally distributed, but also functioned as a learning trial for the participants to orient themselves, and only the second and third trials of the procedure were used in the analysis.

This procedure was chosen to replace an ANOVA as the basic assumption of an ANOVA was violated, namely that there was more than one test or trial per subject (Huck, 2004). The repeated measures ANOVA falls within a mixed model as the three trials on the KFA task violate the assumption of a linear model, which states that errors

are random. In addition, the subjects were matched in the previous study and this study, thus violating the assumption of independence (Huck, 2004). However, the limitation of this method is that outliers from the mean can adversely affect the findings and thus obscure the differences between groups.

Chi-square tests and follow-up analysis

The scores for both the original KFA task and the self-adapted KFA task were used to group the participants into three categories, namely those whose scores increased after interpolation, those whose scores remained the same after interpolation and those whose scores decreased after interpolation. Due to the fact that these three categories represent nominal data, chi-square tests were calculated, according to the group that the participants were in (control or residents) (Huck, 2004). A chi-square test is an inferential test, which involves a critical value that is pulled from, and a *p*-value that is tied to, one of the chi square distributions (Huck, 2004). The chi-square procedure was used to determine whether the scores obtained by the two groups on both the original and self-adapted KFA task were at all associated with groups. That is, a chi-square test would indicate whether the scores obtained on the original and self-adapted KFA are at all related to the group of schizophrenic patients or controls, and thus is an important procedure in terms of the central focus of this particular study.

A follow-up analysis was done using a paired samples t-test. This test was conducted using the actual numerical difference between the second and third trial of the shortened version of the KFA task for both the control and schizophrenic patient group. This was done in order to explore the extent of the difference between the scores of the two groups (that is, the extent of over- or underestimation), as the chi-square test mentioned above merely utilised the assigned categories, which does not reveal the extent of the differences in estimation between the two groups. A paired

samples t-test was selected as the two groups were matched on a number of variables, as mentioned previously, and the second and third trials of the shortened version of the KFA task were normally distributed, thus meeting parametric assumptions.

Mixed ANOVA

In order to address the third research question, a mixed ANOVA was conducted using the data obtained from cohort 2 only for both the shortened version of the KFA task and the self-adapted KFA. This was done in order to determine whether the pattern of scores obtained by the control group and group of schizophrenic patients differed according to which KFA task they completed, and thus provided more information about the reliability of the self-adapted KFA task. This measure was also useful in terms of further exploring the estimation patterns of the two groups across both KFA tasks.

Chapter three
Results

Summary statistics

The mean age of the participants in this study was 44.9 years (N=64, M=44.9, SD = 11.94), with more male (N=36, 56.25%) than female (N=28, 43.75%) participants. Of the 64 participants, 60 were white (93.75%) and 4 were black (6.25%). Fifty-eight participants were right-handed (90.63%), while 6 participants were left-handed (9.37%). The correct estimate in the KFA task was 13 (interval scale scores). The control group, consisting of mentally healthy individuals, scored an average of 15.52 (M = 15.52) across all three of the trials, while the group of individuals with schizophrenia scored an average of 17.38 (M = 17.38) across the three trials.

A Kolmogorov-Smirnov test indicated that the scores for the first trial of the KFA task were not normally distributed ($p < .05$). However, the scores for the second and third trials of the KFA task were normally distributed ($p > .05$). Figure two and three below indicate the accurate shift in the scores that occurred across the second and third trials of the original KFA, as opposed to the scores obtained in the first trial, as indicated in figure one. In the first trial, participants scored an average 16.57 (M = 16.57, SD = 3.45) and obtained a range of 18.5. In the second trial, participants scored an average of 16.24 (M = 16.24, SD = 3.15) within a range of 16 and in the third trial they scored an average of 16.54 (M = 16.54, SD = 3.15) within a range of 15, indicating an accurate shift in their scores.

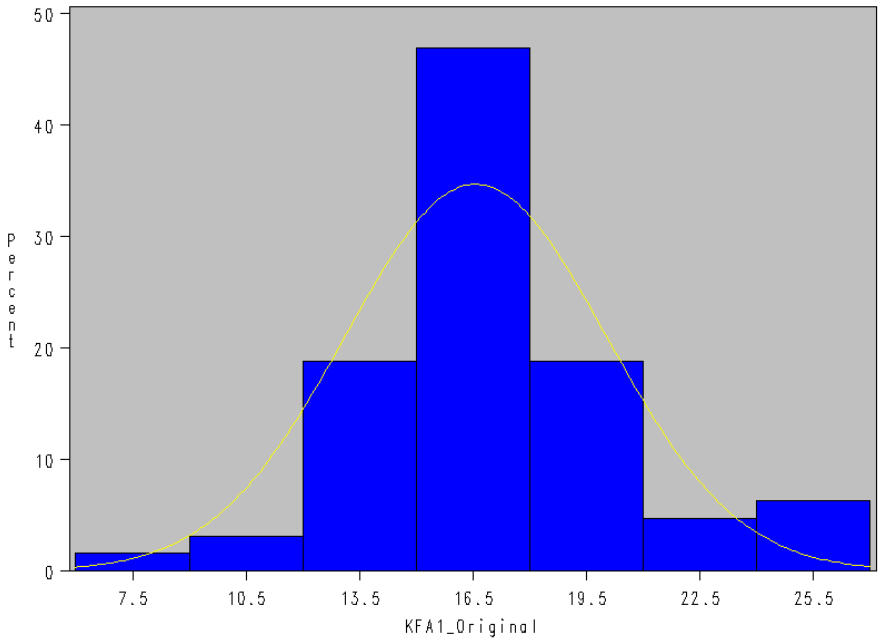

Figure 1: Histogram depicting distribution for the first trial of the original KFA

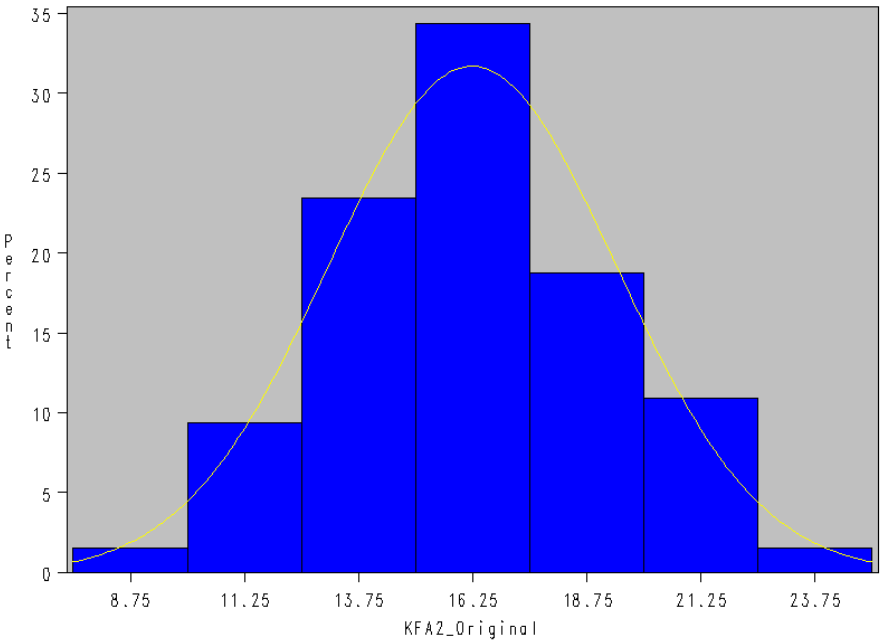

Figure 2: Histogram depicting distribution for the second trial of the original KFA

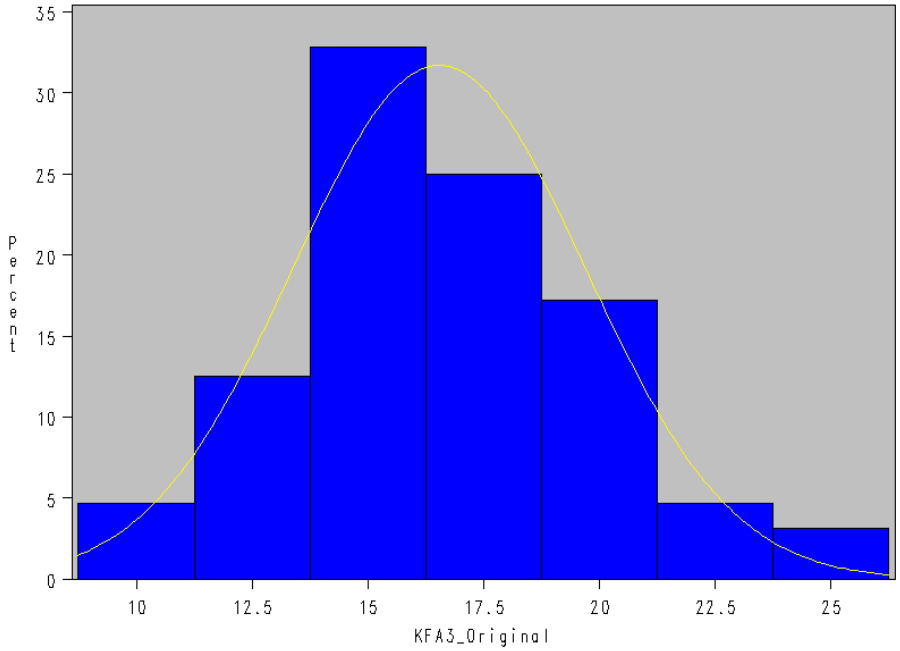

Figure 3: Histogram depicting distribution for the third trial of the original KFA

The correct estimate for the self-adapted KFA task was 16. The control group scored an average of 18.49 (M = 18.49) across all three trials, while the group of schizophrenic individuals scored an average of 19.88 (M = 19.88) across the three trials. A Kolmogorov-Smirnov test indicated that all of the scores for the self-adapted instrument were normally distributed ($p > .05$). The shift between the scores on the three trials of the self-adapted KFA was different to that of the original KFA scores, as indicated by figure four, five and six below. The scores underwent an accurate shift and then became less accurate after interpolation. In the first trial of the self-adapted KFA, participants scored an average of 19.29 (M = 19.29, SD = 2.6) with a range of 10.5. In the second trial, participants scored an average of 19 (M = 19, SD = 3.17) with a range of 12.75, while in the third trial participants scored an average of 19.31 (M = 19.31, SD = 2.88) with a range of 11.75.

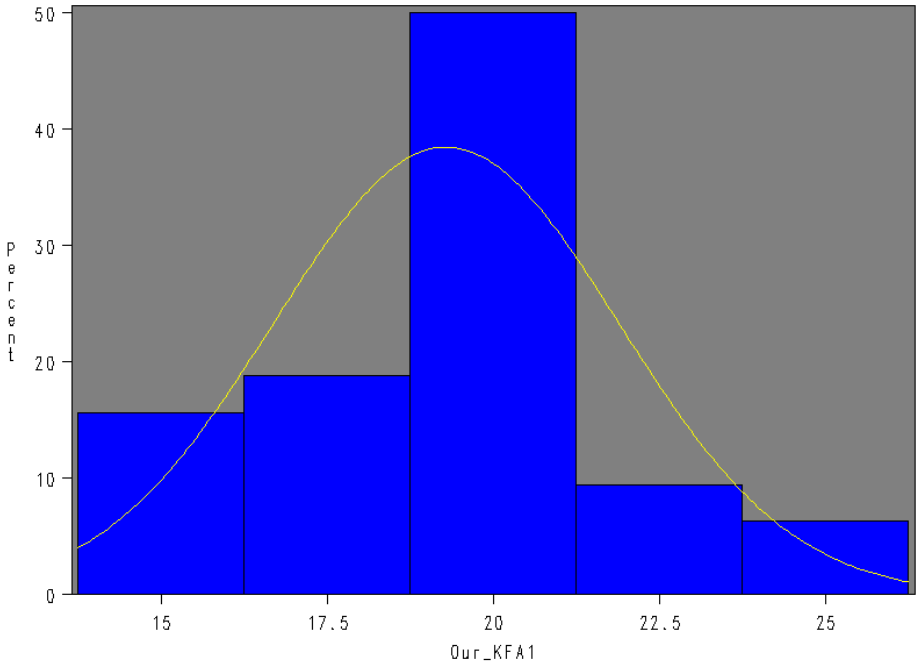

Figure 4: Histogram depicting distribution for the first trial of the self-adapted KFA

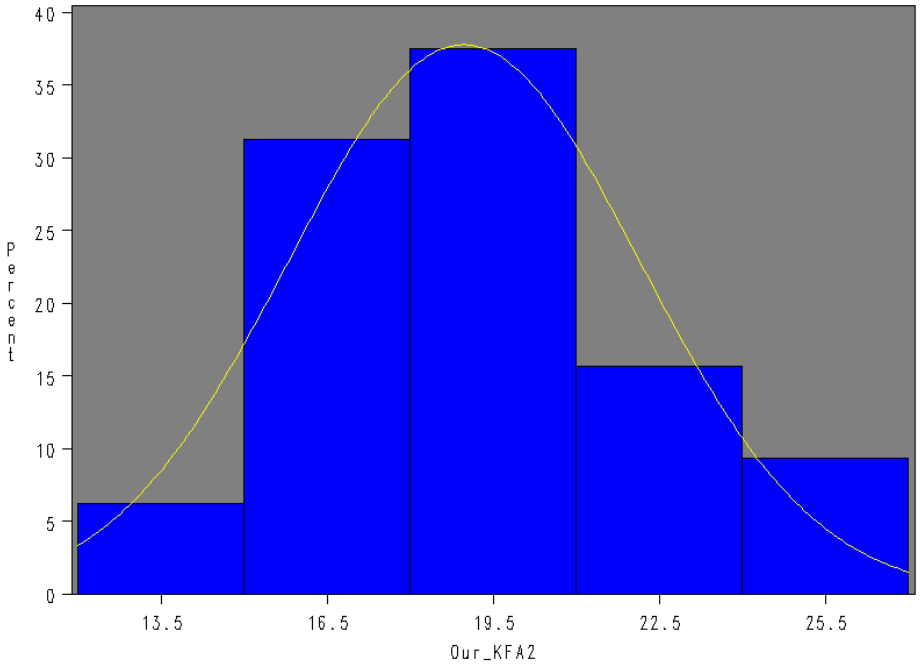

Figure 5: Histogram depicting distribution for the second trial of the self-adapted KFA

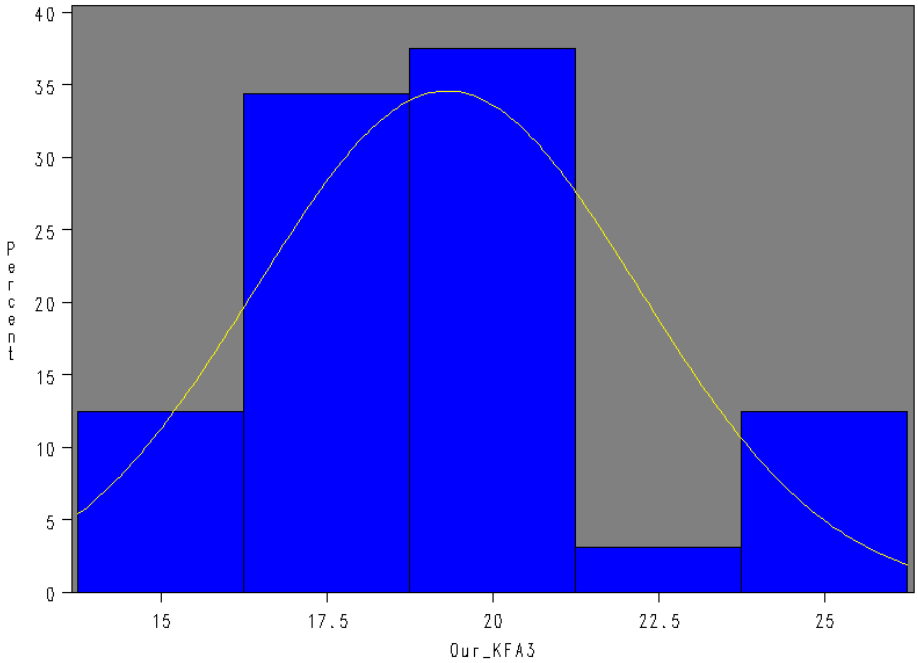

Figure 6: Histogram depicting distribution for the third trial of the self-adapted KFA

Reliability

Due to the fact that the first trial of the KFA task was not normally distributed, it could be argued that the KFA task did not meet parametric assumptions and thus a Spearman's rho correlation was conducted to assess the reliability of the instrument, using the raw scores from the three trials (Neale & Liebert, 1986). The first and second trials of the KFA were strongly correlated ($r = .79$, $p < .0001$), as were the first and third trial ($r = .71$, $p < .0001$). Due to the fact that the second and third trials of the KFA did meet parametric assumptions, a Pearson's product-moment correlation was conducted, indicating that the two trials were strongly correlated ($r = .79$, $p < .0001$). The self-adapted KFA met all of the criteria for a parametric test (at least interval dependent variable, random independent sampling, normally distributed scores, homogeneity of variance and additive means) and a Pearson's product-moment correlation was therefore conducted to establish whether the three trials of the test were correlated (Neale & Liebert, 1986). The first and second trials were strongly correlated

(r = .69, p < .0001), while the first and third trial correlated less strongly (r = .59, p = .0004). The second and third trial of the self-adapted KFA correlated very strongly (r = .79, p < .0001). Thus, there is some evidence for suggesting that both the original KFA task and the self-adapted KFA task designed for the purposes of this study can be said to be reliable measures.

The Pearson's product-moment correlation conducted to determine the test-retest reliability of the self-adapted KFA task yielded a non-significant result, as well as a poor correlation (r = -.15, p = .40) between the two KFA tasks in this study, thus indicating a poor test-retest reliability for the self-adapted KFA task.

Control measures

Age was weakly correlated with the original KFA task as indicated by a Spearman's rho analysis, with the first trial of the KFA correlated positively, but weakly, with age (r = .02, p = .87). The second trial of the KFA correlated negatively, but weakly, with age (r = -.04, p = .73), as did the third trial (r = -.07, p = .55). A Pearson's product-moment correlation conducted with the self-adapted KFA task and age indicated that the first trial of the self-adapted KFA task was negatively and weakly correlated with age (r = -.18, p = .31). The second trial of the self-adapted KFA task was also weakly correlated to age (r = .006, p = .97), as was the third trial (r = .05, p = .77). Thus, given these very low correlations, it is suggested that age did not influence the performance of the participants on the two instruments in any significant way.

A Spearman's rho analysis was conducted in order to determine whether gender correlated with either of the two KFA procedures. All three trails of the original KFA task correlated positively, but weakly with gender. The first trial correlated weakly

with gender (r = .12, p = .34), as did the second trial (r = .19, p = .12), while the third trial correlated the weakest with gender out of all three trials (r = .07, p = .56). The same results were found for the self-adapted KFA task, where all three trials correlated positively and weakly with gender. The first trial correlated weakly with gender (r = .13, p = .45), as did the second trial (r = .14, p = .42) and the third trial, which had the weakest correlation with gender (r = .09, p = .58). Given these very low correlations, it is suggested that gender did not influence the performance of the participants on the two KFA tasks in any significant way.

Cohort effects

As mentioned previously, cohort one consisted of participants that were tested in the first study conducted two years ago, while cohort two consists of participants tested in the present study. The cohorts were compared in terms of age and gender in order to determine whether they differed significantly. A two independent sample t-test was conducted in order to compare the age between the two groups of the two cohorts, as age is classified as an interval scale variable and was found to be normally distributed using the Kolmogorov-Smirnov procedure (p > .15). In the control group, the two cohorts did not differ significantly in terms of age (t_{30} = 1.50, p = .14), although the participants in cohort one were slightly older (M = 48.06, SD = 11.44) than the participants in cohort two (M = 41.82, SD = 12.07). However, the group consisting of individuals with schizophrenia differed significantly in terms of age (t_{30} = 2.56, p = .02), with the participants in cohort one being older (M = 50.12, SD = 10.94) than the participants in cohort two (M = 40.06, SD = 11.25).

Due to the fact that gender is classified as a nominal scale variable, a non-parametric procedure was utilized to compare the cohorts on gender, namely the Mann-Whitney U

test. The participants in the control group did not differ significantly in terms of gender across the two cohorts (Mann-Whitney U = .98, p = .16), with cohort one consisting of less males (seven males) than cohort two (11 males) and more females (eight females) than cohort two (six females). The participants in the group of schizophrenic individuals, on the other hand, differed significantly in terms of gender across the two cohorts (Mann-Whitney U = -2.08, p = .01), with more males in the second cohort (12 males) as compared to the first cohort (6 males) and less females in the second cohort (4 females) as compared to the first cohort (10 females).

With regard to the original KFA task, the control group from cohort one obtained a lower, and more accurate, mean (M = 14.11, SD = 3.14) in terms of the correct estimate on the second trial, than the control group from cohort two (M = 16.22, SD = 2.52). Cohort one of the group of individuals with schizophrenia also scored a lower and more accurate mean (M = 16.71, SD = 3.05) on the second trial of the original KFA than cohort two (M = 17.78, SD = 2.99). With regard to the third trial of the original KFA task, the control group in cohort one were more accurate in their scores (M = 15.13, SD = 3.06) than the control group for cohort two (M = 16.41, SD = 1.79). This was also true for the group of schizophrenic individuals, with cohort one (M = 16.25, SD = 3.18) scoring lower than cohort two (M = 18.28, SD = 3.73).

A two independent sample t-test was conducted on the scores obtained from the second and third trials of the original KFA in order to compare the two groups across the cohorts. With regard to the second trial of the original KFA, the control group for cohort one and two differed significantly in terms of their scores (t_{30} = -2.10, p = .04), with the control group from cohort one scoring lower and more accurately (M = 14.11, SD = 3.14) than the control group from cohort two (M = 16.22, SD = 2.52). However,

the group of schizophrenic individuals did not differ significantly across the two cohorts in terms of their scores for the second trial of the original KFA (t_{30} = -.99, p = .32). In terms of the third trial of the original KFA, the control group for cohort one and two did not differ significantly in their scores (t_{30} = -1.46, p = .15). The same was true for the group of schizophrenic individuals, as the two cohorts did not differ significantly in terms of their scores (t_{30} = -1.66, p = .10).

T-tests

Due to the fact that the group of controls and schizophrenic patients were matched on gender, handedness and to within five years of age, a paired samples t-test was conducted in order to determine whether a difference exists between their mean estimates on each of the three trials of the shortened version of the KFA task. The analysis revealed a significant difference between the two groups on all three of the trials of the shortened version of the KFA task. The group of controls and schizophrenic patients differed significantly in terms of their estimates on the first (t_1 = 27.17, p = .0234), second (t_1 = 29.03, p = .0219) and third (t_1 = 66.38, p = .0096) trial of the shortened version of the KFA task, with the control group obtaining more accurate estimations than the group of schizophrenic patients across each of the three trials.

No significant difference was found for the paired samples t-test conducted between the second and third trials of the shortened version of the KFA task for the entire sample (t_{63} = -1.18, p = .2422). This test was not conducted between the first and second trial of the shortened version of the KFA task, as the first trial served as a learning trial in order for the participants to orient themselves to the task and was therefore not useful in terms of the analysis. A non-significant result was expected due

to the fact that some participants overestimated while others underestimated, thus balancing the scores for the entire sample out.

Repeated measures Analysis of variance

Although the first trial of the original KFA task was not normally distributed and therefore violated the parametric assumptions, a repeated measures ANOVA was conducted using the scores from the second and third trial of the KFA task, which were normally distributed and met parametric assumptions. The repeated measures ANOVA was utilized in the analysis of the scores from the original KFA task in this study. It was conducted specifically to examine the interaction between the two groups and the pattern of change in the mean scores for the second and third trials of the KFA task. This interaction was not significant ($F_{1, 96} = 1{,}18$, $p = .28$), which indicated that the participants' membership to a group, that is, the control group or the group of schizophrenic individuals, did not significantly influence their pattern of scores on the two trials of the KFA in any particular way, although it is noted that the discriminatory power of this analysis is very sensitive to outlying scores.

Chi-square tests and follow-up analysis

The chi-square test was conducted on the original and self-adapted KFA scores, in order to determine whether the scores were associated with the two groups. In the original KFA task, there was a significant difference between the two groups in terms of their scores after interpolation. Eighteen participants (56.25%) and thus the majority of participants in the control group estimated a greater measurement after interpolation, higher than their estimation in the second trial, while seventeen of the participants (53.12%) in the group of schizophrenic individuals estimated a smaller measurement after interpolation, lower than their estimation in the second trial. This

has been illustrated in table 1. This was significant as the chi-square value was 7.3114 with a *p*-value of .0258 ($\chi^2_2 = 7.3114$, $p = .0258$), as indicated in table 2. This result indicates that there is an association between residency (that is, schizophrenic versus mentally healthy) and over- or underestimation on the KFA task after interpolation.

Table 1: Chi-square test for the original KFA procedure

Table of Resident by KFA Difference				
Resident	**KFA Difference**			**Total**
	-1	0	1	
1	8 25.00 32.00	6 18.75 85.71	18 56.25 56.25	32
2	17 53.13 68.00	1 3.13 14.29	14 43.75 43.75	32
Total	25	7	32	64

Table 2: Statistics for Table 1 for the original KFA procedure

Statistic	**DF**	**Value**	**Probability**
Chi-Square	2	7.3114	0.0258
Likelihood Ration Chi-Square	2	7.7776	0.0205
Mantel-Haenszel Chi-Square	1	2.9583	0.0854

A paired samples t-test was then conducted using the numerical difference between the second and third trial for both the control group and group of schizophrenic patients, in order to reveal the extent of the difference between their estimations. The paired samples t-test revealed a significant difference between the two groups ($t_{63} = -4.49$, $p < .0001$), in terms of the change in their estimations from the second to the third trial, thus confirming the significant difference found using the chi-square analysis.

The opposite result was found in the self-adapted KFA task, which was only administered to the second cohort, as compared to the shortened version of the KFA task. That is, ten participants (62.5%) and therefore the majority of the participants in the control group estimated a smaller measurement after interpolation, lower than the estimation made in the second trial, while eleven (68.75%) of the participants in the group of schizophrenic individuals estimated a greater measurement after interpolation, higher than the estimation made in the second trial, as indicated in table 3. This difference was also significant, with a chi-square value of 6.3526 and a *p*-value of .0417 ($\chi^2_2 = 6.3526$, $p = .0417$), illustrated in table 4. This indicates that there is an association between residency and over- or underestimation on the self-developed KFA task. Thus, both of the results indicate an association between residency and the pattern of scores on the instrument, although the two findings occur in opposite directions.

Table 3: Chi-square test for the self-adapted KFA procedure

Table of Resident by Self-adapted KFA Difference				
Resident	**KFA Difference**			**Total**
	-1	0	1	
1	10 62.50 76.92	1 6.25 33.33	5 31.25 31.25	16
2	3 18.75 23.08	2 12.50 66.67	11 68.75 68.75	16
Total	13	3	16	32

Table 4: Statistics for Table 3 for the self-adapted KFA procedure

Statistic	DF	Value	Probability
Chi-Square	2	6.3526	0.0417
Likelihood Ration Chi-Square	2	6.6223	0.0365
Mantel-Haenszel Chi-Square	1	5.7008	0.0170

Mixed ANOVA

In addressing the third research question, a mixed ANOVA was conducted using the data obtained from both the control and schizophrenic patient group in the second cohort, on both the shortened version of and the self-adapted KFA task. This analysis was conducted to explore the reliability of the self-adapted KFA task, as well as to further explore the estimation pattern of the two groups on both KFA tasks. The three variables used in the analysis included two independent variables, namely participant group and type of KFA task, while the dependent variable included the change from the second to the third trial in the KFA tasks. A non-significant result was obtained for both main effects, namely the group effect ($f_{1,61} = 1.70$, $p = .20$) and the KFA task effect ($f_{1,61} = .01$, $p = .91$), indicating no significant difference between the residents or the KFA tasks. However, a significant result was found for the interaction effect ($f_{1,61} = 7.01$, $p = .01$) in that the control groups' scores increased and the group of schizophrenic patients' scores decreased in the shortened version of the KFA task, while the exact opposite was true for the self-adapted KFA task, where the control groups' scores decreased and the group of schizophrenic patients' scores increased. Thus, the analysis indicated that the two KFA tasks were not measuring the same construct.

Chapter four

Discussion

The present study examined whether a shortened version of the KFA task could yield the same internal consistency as the original instrument with a sample of 32 schizophrenic patients and 32 mentally healthy controls. It also examined whether a self-developed component of the KFA could provide further evidence for test-retest reliability for the shortened version of the KFA, conducted with a sample of 16 schizophrenic individuals and 16 controls. Participants in the study were matched on age (to within five years), gender and race across the two cohorts. The control groups within the two cohorts did not differ significantly in terms of age or gender, while the group of schizophrenic individuals differed significantly across the two cohorts, both in terms of gender and age.

Contrary to the findings obtained in the previous study, the present study found a significant difference between the scores obtained for the group of schizophrenic patients and controls after interpolation on the KFA task, as indicated by a chi-square test. This finding is in line with the hypothesised result and confirms the wealth of findings indicating a significant difference between schizophrenic patients and controls in terms of their somatosensory processing, established using the KFA task. In addition, a significant difference was found between the two groups in terms of the estimations on each of the three trials of the shortened version of the KFA.

However, this result should be interpreted with caution following the non-significant result obtained from the repeated measures ANOVA. Due to the fact that the interaction between the two groups and the pattern of change in the mean scores for the

second and third trials of the KFA task was found to be non-significant, it could be argued that no significant difference exists between the two means. This would then imply that no real difference exists between the scores obtained by the two groups on the KFA task.

There may, however, be several reasons that could explain why a non-significant difference was found by the repeated measures ANOVA, while the chi-square test yielded a significant difference between the two groups. The first reason concerns the nature of the repeated measures ANOVA procedure. That is that the repeated measures ANOVA is sensitive to outlying scores, which influence the mean in a great way by distorting it (Huck, 2004). Another reason that could explain why a significant difference was found using the chi-square test and not the repeated measures ANOVA, is that the chi-square measures each score within a category, such as a larger or smaller estimate on the KFA. However, the actual difference in the means could be very small but each score is only placed within one category and as such, the chi-square test may emphasise the difference between groups when the means themselves may not be all that different. However, the follow-up paired samples t-test indicated a significant difference between the group of controls and schizophrenic patients, indicating different means. It is important to note that the chi-square is more likely to show the direction in which the scores differed as opposed to the repeated measures ANOVA, which shows the aggregate size of the difference. Thus, the study found that the aggregate means were not significantly far apart between the schizophrenic patients and controls (noting the complicating effect of outliers) but that the group directions their scores moved in were significantly different.

When examining the results of the chi-square test for the original KFA task, it can be seen that the majority of the participants in the control group estimated a greater score after interpolation while the majority of the participants in the group of schizophrenic patients estimated a lower score after interpolation. More participants' scores in the control group remained the same as compared to the group of schizophrenic individuals. Although a shortened version of the KFA task was used, the control group exhibited a shift towards a higher estimation after interpolation, while the group of schizophrenic individuals exhibited a shift towards a lower estimation. This finding is like that of many other studies conducted using the KFA with schizophrenic and healthy participants. Namely, it points to the phenomenon where schizophrenic individuals tend to estimate lower scores on the KFA after interpolation, which has been found in a number of other studies (Mishara et al, 1973; Houpt et al, 1972; Ritzler & Ebner, 1973). Due to the fact that similar results to previous studies were obtained using a shortened version of the KFA task, it points towards the robustness and consistency of this measure of somatosensory processing, as established using the KFA. That is, that even a short version of the KFA can yield a significant and consistent difference in the measurement of somatosensory sensitisation following satiatory stimulation between controls and individuals with schizophrenia.

The augmenting and reducing paradigm that had been used previously to explain such findings is dated and has been criticised on various fronts as lacking validity. However, it may be that the sensory gating paradigm, discussed earlier, may offer some explanation for the abovementioned findings. That is, that research conducted around the 'desensitivity' exhibited by schizophrenic individuals may offer some understanding for these findings. The abovementioned results indicate that somatosensory sensitivity in healthy controls increases following the interpolation

procedure. It may be that this increase in somatosensory sensitivity following satiation reflects ordinary somatosensory processing in mentally healthy individuals. That is, that mentally healthy individuals may become more sensitive to their senses, such as touch, following stimulation, such as rubbing. The opposite is evident in schizophrenic individuals and may be explained by two possible hypotheses. The first hypothesis that could be used to explain why schizophrenic individuals become less sensitive after stimulation is that regarding functional connectivity, discussed earlier. That is the argument that schizophrenic individuals fail to integrate information at higher levels of processing. In other words, the somatosensory stimulation is registered at lower levels of sensory processing in the brain but that this information is not being integrated into conscious awareness, thereby limiting cortical integration, and not causing the sensitisation effect with consciousness (Peled et al, 2001).

The second hypothesis that could potentially explain the findings obtained from the schizophrenic individuals concerns sensory gating. Here it is argued that due to sensory gating failures, schizophrenic individuals may in fact experience an intense response to satiation at lower levels of somatosensory processing, which may result in the 'shutting down' of stimulus intensity at higher levels. In other words, these individuals may exhibit a compensatory mechanism, which prevents them from becoming overwhelmed by this intense response (Buchsbaum & Silverman, 1968).

Although both hypotheses appear credible and may provide insight into somatosensory processing in schizophrenia, more research is needed in order to distinguish between the two positions, as well as to establish which of the two is accurate. However, the majority of the previous studies indicating lower estimates obtained on the KFA task by schizophrenic individuals point towards the second hypothesis, which argues that

because schizophrenic patients experience sensory overload due to sensory gating deficits, they compensate for this by ‘dampening down’ sensory input, which results in them perceiving stimuli at a decreased intensity (Buchsbaum & Silverman, 1968).

However, the findings obtained in the present study could, in fact, be due to methodological issues surrounding the use of a shortened version of the KFA task. That is, the shortened version of the KFA may reduce the capacity of the instrument to demonstrate consistent patterns of somatosensory processing, which may be established by utilising more trials. Another factor that may have influenced the findings of this study is that of a relatively small sample size. Although a smaller sample of schizophrenic individuals was practically necessary for the present study, it may be problematic in terms of validity and generalisability of the findings that this study would be able to yield.

Further, unlike similar studies conducted in this area, the present study did not include schizophrenic patients in the acute phase of the disorder. Many of the studies conducted in this area have sampled hospitalised schizophrenic patients in the acute phase of the disorder. Individuals in the acute phase of schizophrenia have been found to reduce more than schizophrenic individuals who are not in the acute phase (Kuster et al, 1975). The sample for this study consisted of schizophrenic individuals who were not in the acute phase of the disorder and who were relatively functional, and it could thus be argued that the compensatory mechanism mentioned above, may in fact accompany more acute states of psychosis. This may be the reason that a possible compensatory mechanism was less prominent in this sample.

Whether the finding that schizophrenic individuals estimate significantly lower scores on the KFA than healthy controls is better explained by theories of functional connectivity or theories of sensory gating failures, both of these abnormalities occur at higher levels of somatosensory processing. Therefore, the findings of this study may only be explained by processes that occur at higher cortical levels. It is for this reason that the KFA may be useful in answering questions around processing that occurs at higher cortical levels. More research is therefore required in order to connect the findings obtained using the KFA to findings made using other instruments, especially neurofunctional techniques such as the fMRI and EEG. More research is required in order to establish exactly what aspects of higher cortical processing are impacting on the findings obtained, as well as to establish the exact nature of the construct being measured.

It is important to note that the present study developed a self-adapted, repeated measures version of the original KFA in order to establish whether the findings from the original KFA would be replicated using a slightly different instrument. This self-adapted KFA task was found to have poor test-retest reliability when compared to the shortened version of the KFA task. One explanation for this result could be that the participants in the study became more sensitised over time, thus explaining the poor correlation between the two KFA tasks, and indicating that the tasks should possibly be administered on different days in order to eradicate this sensitising effect. In terms of the self-adapted KFA, the chi-square test again yielded a significant result. However, the pattern of results was almost precisely opposite to the pattern found using the original KFA. That is, that the majority of the control group estimated lower scores on the KFA task after interpolation, while the majority of the participants in the group of schizophrenic patients estimated higher scores after interpolation, which was

found to be significant in terms of the mixed ANOVA conducted to explore the estimation patterns between the two groups of both KFA tasks. It therefore has to be considered as to whether these findings potentially undermine the shortened version of the KFA. However, it must be kept in mind that the self-adapted KFA was only used with the second cohort and the findings can therefore not be directly compared to those obtained using the original KFA. In addition, this procedure was administered towards the end of the testing session and the results may have been influenced by other factors related to time and attention. The self-adapted KFA was also administered after a task specifically developed to distract the participants by stimulating their visual sense, where they were told to choose their favourite pictures out of a set of 11 different pictures. However, from the results obtained from the mixed ANOVA mentioned previously, it seems that the two KFA tasks did not measure the same underlying construct and perhaps should be conducted on different days in order to eliminate the sensitising effect of the first KFA task.

The participants in the control group made lower estimates after interpolation and therefore did not show an increased sensitivity to the satiation procedure, as they did with the original KFA. This could be explained by the mechanism of sensory adaptation, which is defined as the change in responsiveness of the sensory system based on the average level of stimulation (Durgin, 2000). However, the finding that the schizophrenic individuals made greater estimates following satiation is more difficult to explain as it seems to contradict the explanatory paradigms provided previously. These findings could seriously undermine the hypotheses provided earlier, which indicates that the issues being discussed may not be fully understood and thus require more research.

On the other hand, it could be that the initial finding that the schizophrenic patients showed increased sensitisation in the self-adapted KFA indicates that they were more comfortable with the research by the time that they performed the second task. In other words, it may take more time (or a reduction in confounding variables present in the testing situation) for schizophrenic participants to become sensitised in the same way that others do. Therefore, it could be that confounding variables, such as anxiety, distraction and unfamiliarity may have influenced the results initially, and that the sensitisation of schizophrenic individuals to somatosensory input may be delayed rather than consistently suppressed or not integrated. However, and as mentioned previously, more research is required in this area of study in order to clarify the construct under examination, with the assistance of other procedures, in order to triangulate findings and steadily develop some construct validity in these ongoing studies. This, in turn, would assist researchers in this area to more accurately conceptualise explanations for the findings obtained thus far.

Thus, future research in this area should aim to examine the influence of confounding variables such as anxiety and unfamiliarity on the performance of schizophrenic patients on the KFA task. Further, similar research should be conducted on different sets of samples, in order to establish whether a pattern can be found in the way that schizophrenic patients estimate scores after interpolation, as compared to healthy controls. In addition, future research in this area should aim to establish the differences between schizophrenic patients in the acute phase of the disorder and those that are not, in terms of their estimates on the KFA. The clarification that the abovementioned studies would bring, would in turn allow for a clarification on the paradigms used to explain this phenomenon.

Reference List

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders (4th ed.) Text Revision*. Washington: American Psychiatric Association.

Apter, J. T. (1981). The "silent" acute abdomen of schizophrenia. *The Journal of the Medical Society of New Jersey*, 78, 679-680.

Baker, N., Adler, L. E., Franks, R. D., Waldo, M., Berry, S., Nagamoto, H., Muckle, A. & Freedman, R. (1987). Neurophysiological assessment of sensory gating in psychiatric inpatients: Comparison between schizophrenia and other diagnoses. *Biological Psychiatry*, 22(5), 603-617.

Bartfai, A., Levander, S. E., Nybäck, H., Berggren, B. M. & Schalling, D. (1985). Smooth pursuit eye tracking, neuropsychological test performance, and computed tomography in schizophrenia. *Psychiatry Research*, 15(1), 49-62.

Bateson, G., Jackson, D., Haley, J. & Weakland, J. (1956). Toward a theory of schizophrenia. *Behavioral Science*, 1(4), 251-264.

Blackwood, D. H. R., Ebmeier, K. P., Muir, W. J., Sharp, C. W., Glabus, M., Walker, M., Souza, V., Dunan, J. R. & Goodwin, G. M. (1994). Correlation of regional blood flow equivalents measured by single photon emission computerized tomography with P300 latency and eye movement abnormality in schizophrenia. Acta Psychiatrica Scandinavica, 90(3), 157-166.

Blumensohn, R., Ringler, D. M. D. & Eli, I. (2002). Pain perception in patients with schizophrenia. *The Journal of Nervous and Mental Disease*, 190, 481-483.

Braus, D. F., Weber-Fahr, W., Tost, H., Ruf, M. & Henn, F. A. (2002). Sensory information processing in neuroleptic-naïve first-episode schizophrenic patients. *Archives of General Psychiatry*, 59, 696-701.

Brixey, S. N., Gallagher, B. J., McFalls, J. A. & Parmelee, L. F. (1993). Gestational

and neonatal factors in the etiology of schizophrenia. *Journal of Clinical Psychology*, 49, 447-456.

Brown, A. S., Begg, M. D., Gravenstein, S., Schaefer, C. A., Wyatt, R. J., Bresnahan, M., Babulas, V. P. & Susser, E. S. (2004). Serologic evidence of prenatal influenza in the etiology of schizophrenia. *Archives of General Psychiatry*, 61(8), 774-780.

Boutros, N. N., Zouridakis, G. & Overall, J. (1991). Replication and extension of P50 findings in schizophrenia. *Clinical Electroencephalography*, 22, 40-45.

Boutros, N. N., Belger, A., Campbell, D., D'Souza, C. & Krystal, J. (1999). Comparison of four components of sensory gating in schizophrenia and normal subjects: A preliminary report. *Psychiatry Research*, 88, 119-130.

Buchsbaum, M. & Silverman, J. (1968). Stimulus intensity control and the cortical evoked response. *Psychosomatic Medicine*, 30, 12-22.

Butler, P. D. & Javitt, D. C. (2005). Early-stage visual processing deficits in schizophrenia. *Current Opinion in Psychiatry*, 18(2), 151-157.

Chang, B. P. & Lenzenweger, M. F. (2001). Somatosensory processing in the biological relatives of schizophrenia patients: A signal detection analysis of two-point discrimination. *Journal of Abnormal Psychology*, 110, 433-442.

Chang, B. P. & Lenzenweger, M. F. (2004). Investigating graphesthesia task performance in the biological relatives of schizophrenia patients. *Schizophrenia Bulletin*, 30, 327-334.

Chang, B. P. & Lenzenweger, M. F. (2005). Somatosensory processing and schizophrenia liability: Proprioception, exteroceptive sensitivity and graphesthesia performance in the biological relatives of schizophrenia patients. *Journal of Abnormal Psychology*, 114(1), 85-95.

Clementz, B. A., Geyer, M. A. & Braff, D. L. (1998). Poor P50 suppression among

schizophrenia patients and their first-degree biological relatives. *American Journal of Psychiatry*, 155, 1691-1694.

Clementz, B. A. & Blumenfeld, L. D. (2001). Multichannel electroencephalographic assessment of auditory evoked response suppression in schizophrenia. *Experimental Brain Research*, 139(4), 377-390.

Cutting, J. (1985). *The psychology of schizophrenia*. New York: Churchill Livingstone.

Durgin, F. H. (2000). Sensory adaptation. In A. Kazdin (Ed.), *Encyclopedia of Psychology*. New York: American Psychological Association and Oxford University Press.

Dworkin, R. H. (1994). Pain insensitivity in schizophrenia: A neglected phenomenon and some implications. *Schizophrenia Bulletin*, 20, 235-248.

Fishbain, D. A. (1982). Pain insensitivity in psychosis. *Annals of Emergency Medicine*, 11, 630-632.

Freedman, R., Adler, L. E., Gerhardt, G. A., Waldo, M., Baker, N., Rose, G. M., Drebing, C., Nagamoto, H., Bickford-Wimer, P. & Franks, R. (1987). Neurobiological studies of sensory gating in schizophrenia. *Schizophrenia Bulletin*, 13(4), 669-678.

Freedman, R., Ross, R. G. & Adler, L. E. (1998). Physiological indicators of the schizophrenia phenotype. In S. J. Watson (Ed.), *Psychopharmacology: 4th Generation of Progress*. United States of America: Lippincott-Raven Press.

Fromm-Reichmann, F. (1948). Notes on the development of treatment of schizophrenics by psychoanalysis and psychotherapy. *Psychiatry*, 11, 263-273.

Gulli, L. F. & Rosick, E. R. (2005). Schizophrenia. In B. Narins (Ed.), *The Gale Encyclopaedia of Genetic Disorders*. Farmington Hills: MI Thomson Gale.

Hajos, M. (2006). Targeting information-processing deficit in schizophrenia: A novel approach to psychotherapeutic drug discovery. *Trends in Pharmacological Sciences*, 27, 391-398.

Hales, R. E., Yudofsky, S. C. & Gabbard, G. O. (2008). *The American Psychiatric Publishing Textbook of Psychiatry (5th ed.)*. Virginia: American Psychiatric Publishing.

Hooley, J. M. & Delgado, M. (2001). Pain insensitivity in the relatives of schizophrenics. *Schizophrenia Research*, 47(2-3), 265-273.

Holzman, P. S., Proctor, L. R., Levy, D. L., Yasillo, N. J., Meltzer, H. Y. & Hurt, S. W. (1974). Eye-tracking dysfunctions in schizophrenic patients and their relatives. *Archives of General Psychiatry*, 31(2), 143-151.

Houpt, J. L., Tucker, G. J. & Harrow, M. (1972). Disordered cognition and stimulus processing. *American Journal of Psychiatry*, 128(12), 1505-1510.

Howe, G. (1991). *The Reality of Schizophrenia*. London: Faber & Faber Limited.

Hsieh, M. H., Liu, K., Liu, S., Chiu, M., Hwu, H. & Chen, A. C. N. (2004). Memory impairment and auditory evoked potential gating deficit in schizophrenia. *Psychiatry Research: Neuroimaging*, 130, 161-169.

Huck, S. W. (2004). Reading statistics and research (4th ed.). Boston: Pearson Education Inc.

Hugo, C. J., Boshoff, D. E. L., Traut, A., Zungu-Dirwayi, N. & Stein, D. J. (2003). Community attitudes toward and knowledge of mental illness in South Africa. *Social Psychiatry Psychiatric Epidemiology*, 38, 715-719.

Iacono, W. G., Moreau, M., Beiser, M., Fleming, J. & Lin, T. Y. (1992). Smooth-pursuit eye tracking in first-episode psychotic patients and their relatives. *Journal of Abnormal Psychology*, 101(1), 104-116.

International Statistical Classification of Diseases and Related Health Problems, 10th

Revision, version for 2007 (Chapter V: Mental and Behavioural Disorders). http://www.who.int/classifications/apps/icd/icd10online.

Johnson, S. C., Lowery, N., Kohler, C. & Turetsky, B. I. (2005). Global-local visual processing in schizophrenia: Evidence for an early visual processing deficit. *Biological Psychiatry*, 58, 937-946.

Jin, Y., Bunney, W. E., Hetrick, W. P., Sandman, C., Patterson, J. & Potkin, S. G. (1997). P50 and experience of hypersensitivity to external stimuli. *Schizophrenia Research*, 24, 233.

Jin, Y., Bunney, W. E., Sandman, C. A., Patterson, J. V., Fleming, K., Moenter, J. R., Kalali, A. H., Hetrick, W. P. & Potkin, S. G. (1998). Is P50 suppression a measure of sensory gating in schizophrenia?. *Biological Psychiatry*, 43, 873-878.

Judd, L. L., McAdams, L., Budnick, B. & Braff, D. L. (1992). Sensory gating deficits in schizophrenia: New results. *American Journal of Psychiatry*, 149, 488-493.

Kaplan, H. I., Sadock, B. J. & Grebb, J. A. (1994). *Synopsis of Psychiatry: Behavioural Sciences/Clinical Psychiatry (7th ed.)*. Maryland: Williams & Wilkins.

Kiehl, K. A. & Liddle, P. F. (2001). An event-related functional magnetic resonance imaging study of an auditory oddball task in schizophrenia. *Schizophrenia Research*, 48, 159-171.

Kircher, T. T. J., Rapp, A., Grodd, W., Buchkremer, G., Weiskopf, N., Lutzenberger, W., Ackermann, H. & Mathiak, K. (2004). Mismatch negativity responses in schizophrenia: A combined fMRI and whole-head MEG study. *American Journal of Psychiatry*, 161, 294-304.

Krishnan, G. P., Vohs, J. L., Hetrick, W. P., Carroll, C. A., Shekhar, A., Bockbrader,

M. A. & O'Donnell, B. F. (2005). Steady state visual evoked potential abnormalities in schizophrenia. *Clinical Neurophysiology*, 116, 614-624.

Kudoh, A., Ishihara, H. & Matsuki, A. (2000). Current perception thresholds and post-op pain in schizophrenic patients. *Regional Anesthesia and Pain Medicine*, 25, 475-479.

Kuster, G., Harrow, M. & Tucker, G. (1975). Kinesthetic Figural Aftereffects in acute schizophrenia: A style of processing stimuli? *Perceptual and Motor Skills*, 41, 451-458.

Levy, D. L., Holzman, P. S., Matthysse, S. & Mendell, N. R. (1994). Eye tracking and schizophrenia: A selective review. *Schizophrenia Bulletin*, 20(1), 47-62.

Luria, A. R. (1976). *The working brain: An introduction to neuropsychology*. London: The Penguin Press.

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, 17, 827-838.

Mishara, B. L., Baker, A. H. & Parker, L. (1973). Kinesthetic Figural Aftereffects: Norms from four samples, and a comparison of methods for classifying augmenters, moderates, and reducers. *Perceptual and Motor Skills*, 37, 315-325.

Neale, J. & Liebert, R. (1986). *Science and behaviour: An introduction to the methods of research*. Englewood Cliffs: Prentice-Hall.

O'Donnell, B. F., Swearer, J. M., Smith, L. T., Nestor, P. G., Shenton, M. E. & McCarley, R. W. (1996). Selective deficits in visual perception and recognition in schizophrenia. *American Journal of Psychiatry*, 153(5), 687-692.

Patterson, J., Jin, Y., Kang, P., Brahman, P. J., Bunney, J., Potkin, S., Sandman, C. & Bunney, W. E. (2003). Sensory gating in schizophrenia: Clinical, P50 and PPI measures. *Schizophrenia Research*, 60, 257-258.

Peled, A., Geva, A. B., Kremen, W. S., Blankfeld, H. M., Esfandiarfard, R. & Nordahl, T. E. (2001). Functional connectivity and working memory in schizophrenia: An EEG study. *International Journal of Neuroscience,* 106, 47-61.

Petrie, A., Collins, W. & Solomon, P. (1958). Pain sensitivity, sensory deprivation and susceptibility to satiation. *Science,* 128, 1431-1433.

Petrie, A., Holland, T. & Wolk, I. (1963). Sensory stimulation causing subdued experience: Audio-analgesia and perceptual augmentation and reduction. *Journal of Nervous and Mental Diseases*, 137, 312-321.

Rado, S. (1960). Theory and therapy: The theory of schizotypal organization and its application to the treatment of decompensated schizotypal behaviour. In S. C. Scher & H. R. Davis (Eds.), *The outpatient treatment of schizophrenia* (pp.87-101). New York: Grune & Stratton.

Ritzler, B. & Ebner, E. (1973). Contrast and stimulus intensity in Kinesthetic Figural Aftereffects with normal and schizophrenic subjects. *Perceptual and Motor Skills*, 37, 927-935.

Ritzler, B. & Rosenbaum, G. (1974). Proprioception in schizophrenia and normals: Effects of stimulus intensity and interstimulus interval. *Journal of Abnormal Psychology*, 83, 106-111.

Ritzler, B. A. (1977). Proprioception and schizophrenia: A replication study with nonschizophrenic patient controls. *Journal of Abnormal Psychology*, 86(5), 501-509.

Rosenthal, S. H., Porter, K. A. & Coffey, G. (1990). Pain insensitivity in schizophrenia: Case report and review of the literature. *General Hospital Psychiatry*, 12, 319-322.

Sadock, B. J. & Sadock, V. A. (2004). *Kaplan and Sadock's Comprehensive*

Textbook of Psychiatry (8th ed.). Maryland: Lippincott Williams & Wilkins.

Sales, S. M. & Throop, W. F. (1972). Relationship between kinesthetic aftereffects and "strength of the nervous system". *Psychophysiology*, 9, 492-497.

Schooler, C., Buchsbaum, M. S. & Carpenter, W. T. (1976). Evoked response and kinesthetic measures of augmenting/reducing in schizophrenics: Replications and extensions. *The Journal of Nervous and Mental Disease*, 163, 221-232.

Sereno, A. & Holzman, P. (1995). Antisaccades and smooth pursuit eye movements in schizophrenia. *Biological Psychiatry*, 37(6), 394-401.

Siegel, C., Waldo, M., Mizner, G., Adler, L. E. & Freedman, R. (1984). Deficits in sensory gating in schizophrenic patients and their relatives. *Archives of General Psychiatry*, 41(6), 607-612.

Singh, M. K., Giles, L. L. & Nasrallah, H. A. (2006). Pain insensitivity in schizophrenia: Trait or state marker?. *Journal of Psychiatric Practice*, 12(2), 90-102.

Sonder, S. (1955). Perceptual tests and acute and chronic status as predictors of improvement in psychotic patients. *Journal of Consulting Psychology*, 19, 387-392.

Spyrelis, A. (2008). Somatosensory processing in schizophrenic and normal individuals across three psychophysical measures. Unpublished Honours Dissertation. University of the Witwatersrand, Johannesburg.

Susser, E., Neugebauer, R., Hoek, H. W., Brown, A. S., Lin, S., Labovitz, D. & Gorman, J. M. (1996). Schizophrenia after prenatal famine: Further evidence. *Archives of General Psychiatry*, 53(1), 25-31.

Taylor, S. F., Tandon, R. & Koeppe, R. A. (1997). PET study of greater visual activation in schizophrenia. *American Journal of Psychiatry*, 154(9), 1296-1298.

Trump, L. & Hugo, C. (2006). The barriers preventing effective treatment of South African patients with mental health problems by The South African Depression and Anxiety Group (SADAG). *South African Psychiatry Review*, 9, 249-260.

Varsamis, J. & Adamson, J. D. (1976). Somatic symptoms in schizophrenia. *The Canadian Psychiatric Association Journal*, 21, 1-6.

Wible, C. G., Kubicki, M., Yoo, S. S., Kacher, D. F., Salisbury, D. F., Anderson, M. C., Shenton, M. E., Hirayasu, Y., Kikinis, R., Jolesz, F. A. & McCarley, R. W. (2001). A functional magnetic resonance imaging study of auditory mismatch in schizophrenia. *American Journal of Psychiatry*, 158, 938-943.

Wu, E. Q., Birnbaum, H. G., Shi, L., Ball, D. E., Kessler, R. C., Moulis, M. & Aggarwal, J. (2005). The economic burden of schizophrenia in the United States in 2002. *The Journal of Clinical Psychiatry*, 66 (9), 1122-1129.

Yee, C. M., Nuechterlein, K. H., Morris, S. E. & White, P. M. (1998). P50 suppression in recent-onset schizophrenia: Clinical correlates and risperidone effects. *Journal of Abnormal Psychology*, 107(4), 691-698.